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Three Essays On Environmental Economics

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Three Essays on Environmental Economics

by

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I dedicate this work to Marlo Ann, my favorite person.

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Three Essays On Environmental Economics

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In Chapter I, a two capital stock adjustment cost model is used to find the effects on productive capital investment of environmental regulation, which forces firms to invest in pollution control equipment. The empirical results show that, for most industries during the 1970's and 1980's, abatement investment stimulates productive investment. This suggests economies of scope between the two types of capital.

The result is surprising in light of general equilibrium effects, as well as the naïve view of adjustment costs, that would cause investment in abatement equipment to have an unambiguously negative effect on investment in productive capital. The results have implications beyond the scope of this paper. Finding economies of scope in other investment pairs will have strong implications when it comes to government policy regarding the control of the composition of total investment.

Not all forest amenities are derived from rotation length. Chapter II extends current forestry economics literature by allowing firms to optimize both over rotation time and harvest density. I use a two-part tax instrument, a "clear-cut" tax combined with a lump sum licensing fee, to correct for market inefficiency. The sign and magnitude of the taxes depends upon the externality. In general, the licensing fee can optimally take the form of either a tax or a subsidy. A stylized numerical model shows a case where a clear-cut tax can be used with a licensing subsidy to correct for market inefficiency.

In Chapter III risk assessment and risk management aspects of an environmental health problem are fused. Damages from fetal Bisphenol-A exposure are quantified using risk assessment methods. The variability and uncertainty are tracked throughout the entire model. The final distribution reflects variation in exposure, uncertainty and variability in dose response, and in the estimates of the economic costs of the outcomes. The methodology can be extended to the assessment of other harmful environmental chemicals.

The expected damages from future cancers due to fetal BPA exposure are surprisingly small. However, the estimated distribution is not narrow. This model demonstrates the type of regulatory benefits analysis on which policy regulating environmental chemicals should be based.

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Introduction

This dissertation delves into a wide variety of topics including investment and abatement, forest thinning and taxes, and environmental estrogens and risk assessment. In doing so, it uses a wide variety of economic techniques: theoretical, econometric, computational, and benefit/cost analyses.

In Chapter I, "Environmental Regulation: Does Federally Mandated Pollution Abatement Investment Lead to Less Productive Investment?" I measure the correlation between a firm's investment in pollution abatement equipment and its investment in other types of capital.

Environmental regulation, its effectiveness, and its effects on output have been hot topics for the last thirty years. With increasing environmental awareness in the late 60's and early 70's, the number of federal environmental regulations grew rapidly. As part of the new regulation, firms were often required to invest in otherwise unproductive pollution abatement capital. Some industries were hit particularly hard. For instance, in 1973 pollution abatement investment constituted almost thirty percent of the petroleum refining industry's total investment. In the primary metals and paper industries, more than twenty percent of total investment went to the reduction of pollution. At the same time that environmental regulation was growing, the U.S. saw a slowdown in productivity growth and productive capital formation. Some implicitly blame the

regulation for crowding out productive investment. Others say the regulation was insignificant in the economic slowdown.

This paper empirically tests whether pollution abatement investment actually crowds out productive investment. I use an adjustment cost model that allows firms to invest in two different types of capital. For the U.S., between 1973 and 1993, I am able to reject crowding out at the two-digit industry level. Furthermore, I find a positive correlation between productive and abatement investment, suggesting economies of scope between the two.

In Chapter II, "Considering Harvest Percentage in a Forest with Standing Value: Capturing External Benefits Derived from Forest Density", I analyze a Faustian forestry model where the logging firm can choose what percentage of each acre it wishes to harvest.

Over 150 years ago, Martin Faustmann (1849) contributed what would be the key model and starting point for any study of multiple-period, optimal forest rotation. Just within the last 30 years, in the shadow of declining quality forest area and increased conservation efforts, much has been written pertaining to optimal forest rotation. However, very little attention has been given to the optimal harvest percentage per acre.

This chapter extends current forestry literature by allowing the forest planner and private

landowner to choose not only the rotation time, but also how many trees per acre to cut. Many forest amenities are derived not only from the age of the trees, but also from the density of the trees. In the case of some forest externalities such as erosion control, it is possible that the clear-cutting of a forest might result in much larger damages than would occur with selective cutting.

The regulation of the percentage of trees cut per acre may dampen some of the externality paths and thus avert such disasters as the one experienced in Portland, Oregon in 1995. Massive landslides, occurring mostly in areas of clear-cutting, contaminated the entire city's water supply and caused millions of dollars of damage. The damage was so great that in a recent election, Oregonians were asked to vote on a referendum that would have banned clear-cutting completely. Furthermore, the immense, costly forest fires occurring all over the Western U.S. in 2002 make it obvious that letting forests grow without management can be as bad as clear-cutting. Therefore, on forest lands that are in danger of falling victim to a wildfire and would not otherwise be logged, it may be prudent to provide a firm with incentive to harvest some percentage significantly greater than zero. In either case, the consideration of harvest percentage is an important part of forest management and has been ignored in the forest economics literature thus far.

This paper focuses on the case where a logging firm does not internalize the benefits from leaving some trees uncut and therefore chooses to clear-cut. I present a pair of tax instruments that could optimally control clear-cutting rather than banning it completely.

In Chapter III, "A Risk Assessment of Bisphenol-A: Accounting for Medical Uncertainty and Data Limitations in Environmental Health Problems", I estimate the damages due to the unregulated use of Bisphenol-A, an endocrine disrupter found in polycarbonate plastics, dental fillings, the linings of aluminum and tin cans, and in contaminated water supplies. Endocrine disrupters--synthetic chemicals that mimic the effects of human hormones, particularly estrogen--can cause fetal deformities, cancer, and can reduce sperm production. Estimating the possible damages is important to our current government policy, but is also an interesting and formidable economic problem due to the uncertainty arising in toxicology.

Many problems in environmental economics deal with carcinogenic chemicals and the medical uncertainty that comes with them. And though the science is still uncertain, Congress has already passed three laws, the Food Quality Protection Act (1996), the Safe Drinking Water Act (1996), and the Hormone Disruption Research Act (2002), aimed at dealing with endocrine disruptors. On one hand, this legislation is important in safeguarding the public health. On the other hand, as chemical firms are quick to emphasize, these chemicals are very useful, and we should be very careful in passing legislation that regulates or prohibits them.

The difficulty arises in estimating the per capita dose-response function. First, all of the pathways that people can be exposed to Bisphenol-A must be considered in order to

estimate per capita dosage properly. Second, research on endocrine disrupters requires clinical trials, where non-human subjects are typically exposed to doses of the chemical that are much higher than humans encounter in the environment. Furthermore, current research on Bisphenol-A shows that it can alter fetal and child development at very small doses even though negative effects are minimal at high doses.

In this study I use a risk assessment approach to provide a framework for estimating the cost of human consumption of chemicals where the dose-response relationship is uncertain. However, I extend current methodology in several significant ways. First, while risk assessment has come a long way towards quantifying uncertainty and variability in models in past five years, current practice in the field still typically relies on the construction of point estimates, or distributions that rely on at least some point estimates. In this model, I estimate a distribution of damages the chemical may cause. The distribution reflects: (1) uncertainty in the varying doses received by different groups in the population, (2) uncertainty and variability in the physiological response to these doses, and (3) uncertainty and variability in the estimates of the economic costs of these responses. Second, I quantify these uncertainties throughout the model. Furthermore, although risk assessment and risk management are typically thought of separately, this model combines risk assessment and benefit-cost analysis in a framework that can easily be used to analyze the implications of different policies, using current exposure as a baseline. Though steps have already been taken to regulate endocrine disrupters, no such study has been done.

This particular model concentrates on quantifying the health costs of future cancers caused by fetal exposure to BPA *in utero*.

Chapter I
Environmental Regulation: Does Federally Mandated Pollution Abatement Investment Lead to Less Productive Investment?

1. Introductory Remarks and Review of the Relevant Literature

Environmental regulation, its effectiveness, and its effects on output have been hot topics for the last thirty years. With increasing environmental awareness in the late 60's and early 70's, the number of federal environmental regulations grew rapidly. [1] As part of the new regulation, firms were often required to invest in otherwise unproductive pollution abatement capital (e.g. end of pipe abatement devices). Some industries were hit particularly hard. For instance, pollution abatement investment constituted almost thirty percent of the petroleum refining industry's total investment in 1973. In the primary metals and paper industries, more than twenty percent of total investment went to the reduction of pollution.

At the same time environmental regulation was growing, the U.S. saw a slowdown in productivity growth and productive capital formation (Summers, 1981). Some literature has suggested that the slowdown in growth is due to a drop in productive capital formation (Clark, 1978, and Norsworthy, et al., 1979). Many have pointed their fingers at the increase in environmental regulation as the cause of the drop in productive capital

[1] Major legislation: Clean Air Act (1965), National Environmental Policy Act (1970), Amendments to the Clean Air Act (1970), Clean Water Act (1972), Amendments to the Clean Air Act (1977), Amendments to the Clean Water

formation, stating that firms' investment in productive capital is crowded out by the now-mandatory investment in pollution abatement equipment. In accounting for a slowdown in growth due to environmental regulation, some of this literature implicitly assumes that productive investment is crowded out by abatement investment (Denison, 1979, Norsworthy, et al., 1979, Christiansen and Haveman, 1981, Marks, 1991, Collins, 1996). Gray and Shadbegian (1998) test for crowding out in the pulp and paper industry. They find that investment in pollution abatement equipment and productive equipment tends to happen at the same time. However, they find that paper mills that invest in relatively more abatement equipment invest less in productive equipment later on. Porter and van der Linde (1995) suggest that this crowding out was insignificant and was not a factor in the economic slowdown of the 70's.

This paper empirically tests whether pollution abatement investment actually crowds out productive investment. I use an adjustment cost model that allows firms to invest in two different types of capital, an application stemming from a model introduced originally by Wilcoxen (1990). For the U.S., between 1973 and 1993, my results show that the crowding out hypothesis can be rejected for a range of industries. Furthermore I find a positive correlation between productive and abatement investment in some industries, suggesting economies of scope between the two.

Part 2 of this paper discusses the theoretical adjustment cost model in detail, while Part 3 discusses the different possible econometric models implied by the theoretical model.

Part 4 covers the nature and properties of the data used to estimate the model, while Part 5 provides the empirical results and a discussion of the implications.

2. Theoretical Groundwork

This paper generalizes Tobin's (1969) q-theoretic single capital stock adjustment cost model to incorporate two capital stocks (productive capital and pollution abatement capital). The adjustment cost mechanism allows for the possibility of crowding-out or crowding-in of productive investment by abatement investment.

In the q-theoretic model, originally suggested by James Tobin (1969), a firm's rate of investment is a function of "q", where q is the ratio of the market value of new additional capital investment to its replacement cost. When the firm can freely change its capital stock, it will increase or decrease capital investment until q equals one, or equivalently, until the marginal market value of new capital equals the capital's replacement cost. When convex costs of capital installation are assumed, this model is equivalent to a model where adjustment costs are introduced implicitly through a lag function of investment. However, as Hayashi (1982) points out, the q-theoretical model has the advantage in that it allows output to be an endogenous decision.

Thus, when a firm faces convex costs of capital installation, a large investment in pollution control equipment might lead to a decrease in the investment of productive capital by raising its marginal cost. For instance, if adjustment costs arise because

managers become less efficient when faced with large amounts of new investment, then adjustment costs depend on the total amount of investment regardless of the type. In this case, abatement investment could crowd out productive investment on a one-to-one basis. However, full offset is not likely. In general, the adjustment cost function could include economies or diseconomies of scope. If economies of scope exist, then abatement investment could actually stimulate productive investment. Thus, it becomes impossible to determine analytically the effects of pollution abatement investment on the marginal cost of productive capital.

The rest of this section presents a model of optimal investment for multiple capital goods with adjustment costs, followed by the application of the general model to two specific capital stocks: abatement and productive capital.

A profit maximizing firm choosing among n types of capital will solve the following infinite horizon model:

$$\max_{I} V = \int_{0}^{\infty} \{\pi(K, I, W) - P_K I\} e^{-R(t)} dt \tag{1.1}$$

$$\text{s.t:} \qquad \dot{K} = I - \delta K \tag{1.2}$$

Where, K is a vector of n capital stocks, I is the corresponding vector of investment in K, P_K is a vector of purchase prices for capital types at time t, W is a vector of input and output prices at time t, δ is a diagonal matrix of depreciation

rates, $R(t) \equiv \int_0^t r_s ds$ and r_s is the interest rate at time s, $\pi(K,I,W)$ is the firm's profit function, capturing both the rent on capital and internal adjustment costs. [3]

Setting up and differentiating the Hamiltonian, and making use of a transversality condition that requires the current marginal value of capital, λ, to grow more slowly than $r + \delta$, provides the following $3n$ first order conditions for optimality in investment at time zero: [4]

(1.3) $$\lambda = P_K - \nabla_I \pi$$

(1.4) $$\lambda = \int_0^\infty \nabla_K \pi e^{-(R(t)J+\delta t)} dt$$

(1.5) $$\dot{K} = I - \delta K$$

Where ∇_I, ∇_K, denote partial derivatives with respect to multiple investment and capital types, and J is the identity matrix. Intuition for these results are discussed after the following example.

Now that the first-order conditions have been shown for the general, n capital case, these results can be applied to the particular case in this paper. The firm invests in only two

[3] This formulation abstracts from the firm's financial decision regarding the source of funds for new investment. It assumes that investment is funded out of retained earnings rather than by issuing new debt or equity.

types of capital: productive capital (K_p) and pollution abatement capital (K_a). It is assumed, as in past literature (Conrad and Morrison, 1985, and Wilcoxen, 1990), that pollution abatement capital serves only as means of complying with regulation. To the firm, it has no other value. This implies that $\partial\pi/\partial K_a$ is, by definition, equal to zero. From 1.4, it is observed that this also implies that λ_a is zero. So, for example, a firm will not buy a scrubber unless it needs it to comply with regulation. Instead, investment in abatement capital will be treated as exogenously decided by the regulator.[5] This leaves the following set of first order conditions:

$$\lambda_p = P_K^p - \frac{\partial \pi}{\partial I_p} \tag{1.6}$$

$$\lambda_p = \int_0^\infty \frac{\partial \pi}{\partial K_p} e^{-(R(t)J + \delta_p t)} dt \tag{1.7}$$

$$\dot{K}_p = I_p - \delta_p K_p \tag{1.8}$$

From equation 1.7, it is apparent that λ_p can be interpreted as the present value of the sum over time of marginal products accrued by adding a unit of K_p. Furthermore, the right hand side of 1.6 can be interpreted as the marginal cost of investing in an additional unit of productive capital:

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[4] For a detailed description of this process, see Appendix A.

[5] $\dot{K}_a = I_a - \delta_a K_a$ However, since I_a is exogenous, K_a is effectively exogenous.

(1.9) $$mc_p \equiv P_K^p - \frac{\partial \pi}{\partial I_p}$$

With convex adjustment costs, the effect on current profits of an increase in investment ($\partial\pi/\partial I_p$) is negative, so the marginal cost of K_p is greater than its purchase price.

This allows for the isolation of the effect of abatement investment on productive investment. Substituting 1.9 into 1.6 and making use of the implicit function theorem to take the derivative of I_p with respect to I_a produces the following:

(1.10) $$\frac{dI_p}{dI_a} = -\left(\frac{\frac{\partial \lambda_p}{\partial I_a} - \frac{\partial mc_p}{\partial I_a}}{\frac{\partial \lambda_p}{\partial I_p} - \frac{\partial mc_p}{\partial I_p}} \right)$$

Firms are assumed to be price takers, and λ_p is assumed to be independent of investment. That is, λ_p may change over time, but at any given time t, the firm takes it as given.

Thus, 1.10 reduces to the following expression:

(1.11) $$\frac{dI_p}{dI_a} = -\left(\frac{\frac{\partial^2 \pi}{\partial I_p \partial I_a}}{\frac{\partial^2 \pi}{\partial^2 I_p}} \right)$$

As explained earlier, a special case is where a firm's adjustment costs depend only on the total amount of investment, and not on the composition of the investment. In that case, $dI_p/dI_a \equiv -1$ and productive investment is crowded out by abatement investment on a one-to-one basis, as assumed by previous literature. However, a more likely scenario is one where adjustment costs do indeed depend on the composition of the investment, such as through economies of scope in the installation of abatement equipment. This makes both the sign and the magnitude of dI_p/dI_a theoretically indeterminate.

3. The Econometric Model

In order to proceed, a model that can be estimated econometrically must be developed. This forces some assumptions to be made about the form of the firm's profit function. The firm is assumed to be a price taker, to produce under a constant returns to scale production function, and to face adjustment costs in the form of convex installation costs for new capital. Thus the short run profit function can be specified as follows:

$$\pi(K, I, W) = \sigma(W)K_p - \rho(W)\phi(I) \tag{1.12}$$

where $\sigma(W)$ is a homogenous vector function of prices and wages and can be thought of as the marginal returns of each type of capital, $\rho(W)$ is the vector of installation prices of new capital, a homogeneous vector function of prices and wages, $\phi(I)$ is a convex vector function of I that captures the effects of adjustment costs.

As is common in q-theoretic investment literature (Summers, 1981, Blanchard and Fischer, 1989, and Wilcoxen, 1990), I assume that $\phi(I)$ can be approximated by the following quadratic form: [6]

(1.13) $$\phi(I) = \alpha + \gamma' I + I' \Gamma I$$

Where γ is an unknown vector and Γ is an unknown matrix of parameters whose elements correspond to the two types of capital.

Now, substituting 1.12 into 1.1 allows the firm's first order conditions to be rewritten as follows:

(1.14) $$\lambda_p = P_K^p + \rho(W) \frac{\partial \phi(I)}{\partial I_p}$$

(1.15) $$\lambda_p = \int_0^\infty \frac{\partial \pi}{\partial K_p} e^{-(R(t)J + \delta_p t)} dt$$

(1.16) $$\dot{K}_p = I_p - \delta_p K_p$$

Since a functional form for $\phi(I)$ has been assumed, $\partial\phi(I)/\partial Ip$ can be calculated:

[6] Hamermesh and Pfann (1996) suggest that the quadratic form for adjustment costs is an oversimplification, and that "lumpy" or asymmetric functions should be used. For tractability, however, the commonly-used quadratic form is also used here.

(1.17) $$\frac{\partial \phi(I)}{\partial I_p} = \gamma_p + 2\Gamma_{pp} I_p + 2\Gamma_{ap} I_a$$

Where Γ_{pp} and Γ_{ap} are elements of matrix Γ.[7]

Now, substituting 1.17 into 1.14 produces first-order conditions of the form:

(1.18) $$\lambda_p = P_K^p + \rho(W)(\gamma_p + 2\Gamma_{pp} I_p + 2\Gamma_{ap} I_a)$$

(1.19) $$\lambda_p = \int_0^\infty \frac{\partial \pi}{\partial K_p} e^{-(R(t)J + \delta_p t)} dt$$

(1.20) $$\dot{K}_p = I_p - \delta_p K_p$$

Following the previous logic, this implies that the firm's marginal cost of productive investment is:

(1.21) $$mc_p = P_k^p + \rho(W)(\gamma_p + 2\Gamma_{pp} I_p + 2\Gamma_{ap} I_a)$$

Setting marginal cost equal to marginal revenue (λ_p) and solving for I_p yields:

(1.22) $$I_p = \frac{1}{\Gamma_{pp}} \left(\frac{1}{\rho(W)} (\lambda_p - P_k) - \gamma_p \right) - \frac{\Gamma_{ap}}{\Gamma_{pp}} I_a$$

This suggests that the effect of abatement investment on productive investment can be found by estimating the sign and magnitude of Γ_{ap}/Γ_{pp}. This is confirmed by making use of the implicit function theorem on 1.21 to find dI_p/dI_a.

(1.23) $$\frac{dI_p}{dI_a} = -\frac{\Gamma_{ap}}{\Gamma_{pp}}$$

The sign of 1.23 will be telling. Since adjustment costs are assumed to be convex in I_p, the sign of Γ_{pp} will be *a priori* positive. Therefore, Γ_{ap} must be negative if dI_p/dI_a is positive. This could be interpreted as economies of scope in investment costs.

Now, in order to estimate the model, data on I_p, I_a, λ_p, P_k^p, and $\rho(W)$ must be collected. The data for I_p, I_a, and P_k^p are readily available at the two-digit industry level. These data may be used to estimate 1.22 under the assumption that industries may be modelled as representative firms. However, λ_p and $\rho(W)$ are unobservable.

To overcome the problem of an unobservable $\rho(W)$, I assume the price of installation services to be constant relative to the GDP deflator. This allows 1.22 to be rewritten in constant dollar terms for sector i at time t:

[7] For detailed descriptions of this process see, Appendix B.

(1.24) $$I_p^{it} = \frac{1}{\Gamma_{pp}^i}\left(\frac{\lambda_p^t - \left(P_K^P\right)^t}{\rho_0} - \gamma_p^i\right) - \frac{\Gamma_{ap}^i}{\Gamma_{pp}^i} I_p^{it}$$

Where I_p, I_a, λ_p, P_k^p, K and ρ_0 are expressed in constant dollar terms.

To see how λ_p might be measured, past literature can be examined. Hayashi (1982) used a restricted q-theoretic model to show that marginal q is equal to average q, and so subsequent q-theory literature has often used stock market indices as a proxy for marginal q. The model discussed in this paper implies $q_p = \lambda_p / P_k^p$, so a determination of q_p also determines λ_p. However, using stock market indices as a general proxy for q is insufficient for this study. Since the model here has multiple firms and two types of capital, many average q's would need to be calculated. Untangling stock market data to do this is nearly impossible because most firms produce many different types of products.

This suggests that the λ_p in this model must be handled in another manner. Suppose that for a representative firm, or industry i, in year t, λ_p can be decomposed as follows:

(1.25) $$\lambda_p^{it} = \lambda_p^i + \lambda_p^t$$

Where λ_p^i varies across industries and λ_p^t varies across time.

Further, to produce an estimable function, one more assumption must be made. Ideally, given a larger data set, the parameters of $\phi(I)$, α^j, γ^j, and Γ^i would be allowed to vary across industries without restriction. This would be comprable to running OLS on each industry separately. However, to increase the power of estimation, I make the following assumption on how the parameters of the adjustment cost function vary across industries:

(1.26a) $$\alpha^i = \frac{\alpha}{\bar{K}^i}$$

(1.26b) $$\gamma^i = \frac{\gamma}{\bar{K}^i}$$

(1.26c) $$\Gamma^i = \frac{\Gamma}{\bar{K}^i}$$

Where $\bar{K}^i$ is the average capital stock for a firm in industry *i*.

Substituting 1.25 and 1.26 into 1.24 yields the following equation:

(1.27) $$I_p^{it} = -\frac{\gamma_p}{\Gamma_{pp}} + \frac{1}{\rho_0 \Gamma_{pp}} \bar{K}^i \left(\lambda_p^t - (P_k^p)^t \right) + \frac{1}{\rho_0 \Gamma_{pp}} \bar{K}^i \lambda_p^i - \frac{\Gamma_{ap}}{\Gamma_{pp}} I_a^{it}$$

As is now apparent, the assumptions made in equations 1.25 and 1.26 make the empirical testing of the model more feasible in several ways. First, separating λ_p into industry and time components allows the use of stock market data as proxies for time effects in the model. Whereas stock market data are insufficient proxies for *q* when many firms are

represented, it is a suitable proxy for the time component of q. For example, if the Standard & Poor's index is particulary high in one year, it is likely the marginal value of capital is high in that year also. Assuming $\overline{K}$ varies by industry but not by time allows it to be treated as an industry-specific effect. Thus, the industry-specific and time-specific effects on all variables other than I_a are completely captured by running a pooled regression using the Standard & Poor's (S&P) stock market index as a proxy for the time-sensitive variables and including industry dummies. Then the following model correctly captures the overall effect of abatement investment on productive investment:

$$I_p = \beta_0 + \beta_1 I_a + \beta_i' D_I + \beta_{isp}' D_{ISP} + \varepsilon_{it} \tag{1.28}$$

Where D_I is a set of dummy variables for industries, and $D_{ISP} = D_I*(S\&P)_t$ and is a set of interactive dummy variables. The industry dummy is multiplied by the S&P stock index, normalized to 1982 dollars. β_0 is the estimated intercept and does not change relative to industry, time, or amount of abatement investment. β_1 is the effect of abatement investment on productive investment. β_i captures the industry-specific effect relative to the constant. β_{isp} captures the time-specific effects for each industry.

Equation 1.28 captures the common effect of I_a on I_p, but it constrains the effect to be equal across industries and time. It is likely that the effect of I_a is different for each industry. Furthermore, a new-source effect may cause the correlation between I_a and I_p to increase over time. When regulations were first enacted, investment in pollution

abatement equipment was primarily geared toward controlling emissions from sources already in place (and thus may have come at a cost to new productive investment). However, as this backlog is eliminated, and the firm begins to invest in new productive capital, it may find that new productive capital cannot be installed without the proper abatement equipment. This would be expected to cause the correlation between I_a and I_p to increase over time. Up until 1980, nearly all abatement investment was applied to existing sources, so allowing the slope coefficients on I_a to change between the sub-periods 1973-1979 and 1980-1993 can correct for the possible misspecification of 1.28. [8,9, 10] Therfore, in order to capture the different effect I_a has on I_p across industries and across time, this paper also estimates:

$$I_p = \beta_0 + \beta_{1i} I_a D^* + \beta_{2i} ID_{IIa} + \beta_i^{'} D_I + \beta_{isp}^{'} D_{ISP} + \varepsilon \tag{1.29}$$

Where $ID_{IIa} = I_a * D_I$ and is an industry dummy interactive with abatement investment, and $D^* = 1$ for the period 1980-1993. Therefore, the slope coefficient β_{2i} is now relevant for the time period 1973-1979 and β_{1i} picks up the difference in slope in each industry over the later sub-period. Notice also that the base abatement investment variable is dropped. This allows β_{2i} to be an estimate of the actual slope coefficient for each industry from 1973-1979.

[8] The results of the likelihood ratio tests of the various constraints are discussed in the Section 5.
[9] It may be that the correlation is increasing over time due to reasons besides the new source effect.
[10] Cutoffs for every year were tested for a "kink" in the data. D*=1 for 1980-1993 resulted in the highest value for the log likelihood function, making it the correct choice to test the significance of a regime change.

4. The Data

The pollution-abatement and productive capital investment are available for the sixteen manufacturing industries in Table 1.1. The data are measured at the two-digit SIC-code level of aggregation.

The data cover the time periods from 1973 to 1986 and 1988 to 1993 and are from the Census Bureau's "Pollution Abatement Costs and Expenditures" study; data from 1987 is not available.[11] The original data are for total capital investment and pollution-abatement capital investment (by industry and year) in current dollars. Subtracting abatement investment from total investment creates the productive investment series. The data are then converted to constant 1982 dollars using the price deflator for Producer's Durable Equipment.

Figure 1.1 shows the correlation coefficient between productive and abatement investment in each industry. From inspection of the chart, it is immediately apparent that twelve out of the sixteen industries show a positive correlation between abatement and productive investment. Eight of the industries have correlation coefficients greater than 0.5. Although this is far from being a robust statistical test, the chart suggests the possibility of an overall positive correlation between I_a and I_p. However, the difference in correlation coefficients shows that the constrained model is most likely a

misspecification.

Further analysis of the data can be performed by looking at the average amount of abatement investment as a percentage of total investment by each industry in Figure 1.2.

Note several points from Figure 1.2. First, in only five industries (paper, chemicals, petroleum refining, primary metals, and Transportation equipment) is abatement investment a substantial percentage of total investment (more than 6%). Next, the investment activity across industries varies greatly. This could be a possible source of heteroscedasticity. Thus it becomes necessary to look at variance in I_p and I_a across industries. Figure 1.3 shows how variances differ across industries.

It is obvious, on observation of Figure 1.3, that variances, especially in I_p, differ greatly across industries. This suggests that a correction for heteroscedasticity is probably in order. Indeed, using the Cook and Weisberg (1983) test for heteroscedasticity, the null hypothesis that $Var(\varepsilon_{it}) = Var(I_p)^I$ is rejected with more than 99% confidence ($\chi^2(1) = 67.22$).

It is also worthwhile to look at percentage change in productive and abatement investment over time in Figure 1.4. In observing this chart, no obvious pattern of investment growth can be ascertained. That is, abatement investment does not

[11] The Census Bureau did not perform the census in 1987.

necessarily grow in the same years as productive investment. The percentage changes in productive investment and abatement investment actually have opposite sign in 8 out of the 19 years. Productive investment decreases in most years between 1976 and 1983, whereas abatement investment generally increases until 1981. Both types of investment show a significant decrease in 1982 and 1983. Furthermore, both types of investment generally increase between 1984 and 1992, except in 1986 and 1991, when abatement investment decreases. This suggests that if a correlation between I_a and I_p exists, the coefficient does not remain constant over time and may be higher in the later sub-period.

The pooled model has 320 observations. Furthermore, in the two equations, 1.28 and 1.29, 49 and 80 parameters need to be estimated, respectively. It is prudent to worry about the small number of degrees of freedom. With the pooled model, however, I maximize the degrees of freedom for the data that are available. Furthermore, as will be apparent in Section 5, the results are surprisingly robust.

5. Empirical Results and Implications

Equations 1.28 and 1.29 are estimated while correcting for heteroscedasticity[12] using a pooled GLS estimator. Cross-sectional correlations were all assumed to be zero. A likelihood ratio test was performed to test the restrictions imposed in 1.28. The null hypothesis that the effect of abatement investment is the same across industries and the

[12] To correct for the heteroscedasticity, the estimated variances can be written in the general form: $(X'X)^{-1}XWX(X'X)^{-1}$, where $W = \sum_i x_i' \overline{\varepsilon_i} \overline{\varepsilon_i} x_i$

two time periods is rejected with greater than 99% confidence ($\chi^2(31) = 204.83$).[13] The results for 1.29 are shown in Table 1.2.

The dummy variables for industry 22 are omitted to avoid collininearity, so values of the industry dummy coefficients are relative to the constant. A Wald statistic is calculated to test the joint significance of the dummy variables. The null hypothesis that they are not jointly significant is easily rejected ($W \approx \chi^2(31) = 737.08$). Separately, the null hypothesis that the S&P interactive dummies and the industry dummies are each jointly insignificant is also rejected ($W \approx \chi^2(16) = 85.83$, and $W \approx \chi^2(15) = 519.40$, respectively).

As expected due to the new source effect, the coefficient on Ia is increasing significantly for more than half of the industries. Nine (food, paper, printing, chemicals, petroleum, primary metals, machinery, electrical machinery, and instruments) of the sixteen industries have positive, significant β_{1i} coefficients. So, for these industries, the effect of abatement investment on productive investment is significantly greater between 1980 and 1993 than between 1973 and 1979, when abatement investment was typically applied existing sources.

More interestingly, however, is the fact that the coefficient on abatement investment is both positive and significant for three (lumber, chemicals, and primary metals) of the 16

[13] The most general model, with the effect of abatement investment varying over time and industry is also tested against other restricted/nested models: one where the effects vary over time, but not industry, and one where the effects vary by industry, but not time. In both cases, the restrictions are rejected.

industries.[14] This suggests that, in these three industries, rather than crowd out productive investment, investment in pollution control capital actually stimulates it.

To analyze this result, recall that the coefficient on I_a is $-(\Gamma_{ap}/\Gamma_{pp})$. Since the estimated coefficient is both positive and significant, either Γ_{ap} or Γ_{pp} must be negative. As discussed earlier, if adjustment costs are convex in I_p, then Γ_{pp} must be positive. Therefore, Γ_{ap} must be negative. This suggests that it costs less to invest in productive capital and abatement capital together than it does to invest in each separately. Or, equivalently, there are economies of scope when abatement investment and productive investment are done together.

This result, although surprising at first, has a rather intuitive explanation. Suppose that, as is often true, installing pollution-control equipment forces a temporary shutdown of part or all of the facility. Thus the shutdown for installation of abatement capital may also allow productive equipment to be replaced or renovated at a relatively small additional cost. Thus, a firm would want to invest relatively heavily in productive capital whenever it is forced to install pollution abatement equipment.

Only the food and instruments industries have a significantly negative coefficient on

[14] It is worth noting that two of the industries (chemicals and primary metals) with positive, significant I_a coefficients are ones listed earlier as having a significant percentage of their investment in abatement equipment. This might mean that economies of scope are only realized in industries investing heavily in abatement equipment. However, the industry hit hardest by the regulations, and having the highest

abatement investment. Of the remaining 11 industries, 7 have mean coefficients below zero and 4 have mean coefficients above zero. Due to the fact that the data set is relatively small, the standard errors on these estimates large and they cannot be distinguished as significantly different from zero. Figure 1.5 shows where the means and the 95% confidence intervals lie for each industry.

From Table 1.2 and Figure 1.5, it apparent that each industry reacts somewhat differently to federally mandated pollution abatement investment. Surprisingly, there is only evidence of a significantly negative effect on productive investment in 2 out of the sixteen industries. Thus it is now apparent that an a priori assumption of crowding out between abatement and productive investment is incorrect. In fact, in some industries, there is evidence of economies of scope between the two types of investment.

6. Concluding Remarks

The results in this paper show clearly that productive investment is not crowded out by investment in pollution abatement capital in all industries. For the lumber, chemicals, and primary metals industries, in fact, it is safe to say that pollution abatement investment actually stimulates productive investment. This result suggests economies of scope for investing in the two types of capital at the same time. Furthermore, the results give empirical support to the Porter's hypothesis and observation (1995) that firms tend to renovate when faced with environmental regulation.

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percentage of investment in abatement equipment, the petroleum industry, as well as the paper industry,

The results are even stronger in light of the assumption that λ is unaffected by investment in abatement equipment. General equilibrium effects cause both interest rates and the price of capital goods to rise when firms demand abatement capital (Wilcoxen, 1988). These effects unambiguously lead to a reduction in productive investment. Therefore, by ignoring them, the observed results are actually biased away from showing a positive correlation between abatement and productive investment.

Some caveats must be kept in mind. With data aggregated to the 2-digit level, it is impossible to see exactly what is happening at the plant or firm level. Regional factors also cannot be observed. This makes it difficult to say exactly what the mechanism is that drives the correlation. It is possible that industries with a few dirty plants located in heavily regulated areas may have closed those plants and opened new plants in different locations. This would cause a large, positive correlation between abatement and productive investment at the industry level. That phenomenon would be consistent with basic observation that abatement investment may stimulate productive investment but the mechanism would not be economies of scope in investment.

Despite the caveats, the results remain very interesting. At the industry level, abatement policy does not seem to crowd out otherwise productive investment in all industries. On the contrary, abatement investment seems to provide an incentive in some industries (via

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have mean estimates negative and relatively close to zero, but insignificant at the 95% confidence level.

economies of scope) for firms to renovate sooner, and in greater quantities than they normally would. (This does not necessarily imply that individual firms are made better off by regulation, just that in aggregate, their investment rises.) Furthermore, the results show that care must be taken when designing policy affecting a large, aggregate branch of the economy. Since industries react to mandated abatement investment differently, the effect on investment in the productive capital stock of such regulation should be calculated separately for each industry.

These results have implications beyond the range of this paper. Finding economies of scope between other investment pairs will have strong implications for policies designed to alter the composition of total investment. In fact, it would be surprising if other types of investment did not interact in the adjustment cost function.

Chapter II
Considering Harvest Percentage in a Forest with Standing Value: Using Two-Part Tax Instruments to Capture External Benefits Derived from Forest Density and Rotation Time

1. Introductory Remarks and Review of the Relevant Literature

Chapter II extends current forestry literature by allowing the forest planner and private landowner to choose not only the rotation time, but also how many trees per acre to cut. Many forest amenities are derived not only from the age of the trees, but also from the density of the forest. In the case of some forest externalities such as erosion control, it is possible that the clear-cutting of a forest might result in much larger damages than would occur with selective cutting.

The regulation of the percentage of trees cut per acre may dampen some of the externality paths and thus avert such disasters as the one experienced in Portland, Oregon in 1995. Massive landslides, occurring mostly in areas of clear-cutting, contaminated the entire city's water supply and caused millions of dollars of damage. The damage was so great that in a recent election, Oregonians were asked to vote on a referendum that would have banned clear-cutting completely. Furthermore, the immense, costly forest fires occurring all over the Western U.S. in 2002 make it obvious that letting forests grow without management can be as bad as clear-cutting. Therefore, on forest lands that are in

danger of falling victim to a wildfire and would not otherwise be logged, it may be prudent to provide a firm with incentive to harvest some percentage significantly greater than zero. In either case, the consideration of harvest percentage is an important part of forest management and has been ignored by the forest economics literature thus far.

This paper focuses on the case where a logging firm does not internalize the benefits from leaving some trees uncut and therefore chooses to clear-cut. I present a pair of tax instruments that could optimally control clear-cutting rather than banning it completely.

Section 2 of this paper briefly revisits the basic Faustmann and Samuelson models of optimal forest management, then introduces a new model that allows a social planner to choose both the optimal rotation period and the optimal amount of forest thinning. Section 3 sets up the private landowner's problem and includes taxes aimed at controlling both rotation time and thinning. I solve the social and private problems, then solve for the optimal tax instruments. Section 4 shows the results of a numerical analysis on a simple stylized case.

2. Fuastmann, Samuelson, and Beyond

Before jumping into a more general model, this section presents a brief overview of the classical Faustmann and Samuelson models.

2.1 The Faustmann Model

The Faustmann model solves for the optimal rotation period of a forest that will be harvested forever. Initially, the land is bare with timber production as its only use. The landowner chooses the rotation period T to maximize the following private value function:

$$V(T) = \frac{G(T)e^{-rT}}{1-e^{-rT}} \tag{2.1}$$

where $G(T)$ is the net timber value of the stand at time T, and r is the interest rate. The numerator can be thought of as the present net value of the stand that will be harvested at T. The denominator is simply the result of summing over an infinite series of identical rotations.

Taking the derivative with respect to the rotation length and rearranging, the following first order condition is derived:

$$\frac{\dot{G}(T)}{G(T)} = \frac{r}{1-e^{-rT}} \tag{2.2}$$

where the left hand side of the equation is the growth rate of the forest in terms of net value. So a rational landowner will cut down all the trees and replant when the growth

rate equals the interest rate multiplied by a discount factor.

2.2 Samuelson: Forest Planner Problem, Externalities Included

Following Samuelson (1976), I introduce an externalities function, in which the externality varies with the age of the forest, into the forest planner's problem. The social value (*SV*) function can now be written the following way:

$$SV(T) = \frac{\int_0^T e^{-rt} F(t)dt + G(T)e^{-rT}}{1 - e^{-rT}} \tag{2.3}$$

where $G(T)$ is again the net timber value at age T, and $F(t)$ is a function giving the externalities generated by the forest at each point in time. The integral gives the present value of a stream of non-timber benefits coming from the standing forest. Thus I have society's total benefit from the forest, summed over an infinite series of rotations.

The time path of the benefits flowing from a standing forest is controversial. A wide literature assumes $F(t)$ increases with the age of the trees at a decreasing rate (Hartman, 1976, Strang, 1983, and Snyder and Bhattacharyya, 1990). If this is the case, the externality function is monotonically increasing, and it will always be optimal (disregarding the timber value) to let the forest grow. Even though this case may be the most likely, it is not the most general case. Calish et. al. (1978) show that, depending on

the forest, the externality function could take any number of shapes, including a non-monotonic one. Englin and Klan (1990) assume only that $F(t)$ is either increasing at a decreasing rate or increasing and then, at some point, decreasing. In this case, the externality function, at some time t_{Fmax} begins to decrease, and it will be optimal (again disregarding timber value) to harvest the forest at some time less than infinity. Therefore, conceptually, the externality may "favor" either older trees or younger trees. Since this paper's main focus is on thinning practices, and a planner would actually want to force a firm to decrease their rotation period in relatively few cases, I assume $F(t)$ is increasing and concave over the relevant range of time.

Now, maximizing SV in equation 2.3 with respect to the rotation length and rearranging, the first order condition can be written as: [15]

$$\frac{\dot{G(T)}}{G(T)} = \frac{r}{1-e^{-rT}}\left(1+\frac{\int_0^T [F(t)-F(T)]e^{-rt}\,dt}{G(T)}\right) \tag{2.4}$$

Thus, the social planner will allow cutting when the growth rate of the forest in net timber value is equal to a discount factor plus the "externalities balance" divided by

[15] Using 2.4, the second order condition reduces to $\dot{F(T)} + \ddot{G(T)} < r\,\dot{G(T)}$

G(T), the timber value at T.[16] The externalities balance is the present value of the difference between the forest benefits available at the end of the rotation period, $F(T)$, and the stream of benefits available from the growing forest throughout the rotation period. Whether the optimal rotation time is shorter or longer than it would be without externalities depends on the sign of the second term on the right hand side. Furthermore, the sign of this term depends on the time path of the externality function. The externality favors old trees, for example, if there is a positive difference between the amenity value at T and the amenity value at t, for all t, summed over the rotation period. For the purposes of this paper, as stated above, this is the case examined here. I assume that for all $t < T$, $F(T) > F(t)$. This implies that consideration of the externality increases the optimal rotation time.

The private landowner who does not internalize the externality will not choose the socially optimal rotation period. Thus the need for corrective regulation or tax instruments arises.

2.3 Beyond Faustmann and Samuelson: Allowing for Forest Thinning and Rotation Timing

To capture the effects of the density of trees left standing after each rotation, as well as the rotation period on forest amenities, I add two major features to the previous model. First, I allow the forest planner or the private landowner to choose not only when to cut,

[16] Note, at this point, as in previous literature, clear-cutting is assumed.

but also what percentage of trees per acre to harvest. This additional choice is important, since many forest amenities such as erosion control are directly related to the remaining density of the forest. It may not be optimal to let the external benefit reach zero, as would be the case with a clear-cut. This is especially true when the positive externality is control of a potential disaster, such as a flood or massive erosion. Second, the externality function, the private gross revenues and costs, and the timber value of the forest are allowed to be functions of the harvest percentage as well as the forest's age.

The forest planner again maximizes the sum of the timber and non-timber benefits of the forest over an infinite number of identical rotations. The value function can be written as follows:

$$SV(T,H) = \left(\frac{1}{1-e^{-rT}}\right)\left(\int_0^T e^{-rt}F(t,H)dt + G(T,H)e^{-rT}\right) \tag{2.5}$$

Note the difference between this expression and equation 2.4. Now the forest externality is both a function of age t and the percentage of trees harvested per acre H. [17] The path of $F(t,H)$ with respect to t has been discussed in detail above. With respect to H, I assume the external benefit decreases as the harvest percentage per acre increases. [18] The net timber value, $G(T,H)$, is also an increasing function of both T and H. Since the forest planner must now make two choices, the optimization problem produces two first

[17] By definition, $0 \geq H \geq 1$. $H = 1$ would be a clear-cut.

order conditions: one describing the optimal rotation time and the other describing the optimal percentage of trees harvested per acre. These first order conditions are:

$$\frac{1}{G(T,H)}\frac{\partial G(T,H)}{\partial T} = \frac{r}{1-e^{-rT}}\left(1+\frac{\int_0^T (F(t,H)-F(T,H))e^{-rt}\,dt}{G(T,H)}\right) \tag{2.6}$$

$$\frac{\partial G(T,H)}{\partial H}e^{-rT} = -\int_0^T \frac{\partial F(t,H)}{\partial H}e^{-rt}\,dt \tag{2.7}$$

Equation 2.6 gives the conditions for optimal rotation time given a certain percentage of trees cut per acre. The social planner, as in the Samuelson model, will allow harvest when the growth rate of the forest in net timber value is equal to a discount factor plus the externalities balance divided by the timber value at T when the harvest fraction is H.

Equation 2.7 gives the first order condition for the optimal percentage of trees harvested per acre given a certain rotation period T. The social planner will choose H such that the marginal net timber value with respect to H is equal to the marginal value of the lost forest amenity with respect to H. Intuitively, the social benefits of a marginal increase in H must equal the social costs of a marginal increase in H.

[18] Recall that the externality is a positive one, an amenity. As the firm increases H, for example, the chance the forest will *prevent* a landslide decreases.

3. Private Optimization, Taxation, and Socially Optimal Outcomes

This section examines the private logging firm's problem, where the firm faces taxes aimed at controlling both the percentage of trees cut on each acre and rotation time. If it were feasible to implement, a Pigovian tax or subsidy on the externality would be a direct way to force the firm to internalize the externality and could induce optimal behavior in both choices. However, a Pigovian tax or subsidy is not feasible in this case. If the externality in question were erosion control, for example, a tax on the amount of dirt that travels from one acre to the next, or into a stream, is unrealistic. The observation of such an externality, and enforcement of the tax, would be very difficult if not impossible.

It is much simpler to tax the market transactions and suboptimal behavior of which the externality is a symptom. In the following analysis I consider two tax instruments. One is a flat, lump-sum licensing fee (τ^{LS}), collected at harvest time.[19] I also consider an additional "clear-cut" tax (τ^{CC}), assumed to be levied on the firm's fraction of trees harvested per acre. The number of trees that can be planted on an acre is assumed to be fixed.[20] Although not a tax on a direct market transaction, and therefore raising some observation and enforcement problems, the clear-cut tax is still simpler to implement than the Pigovian tax. Enforcing the tax will require someone to physically observe each site

[19] To control for rotation period alone, Englin and Klan suggest the use of existing tax instruments such as a property tax, a severance tax, or a yield tax. While these existing, market-based instruments are interesting and intuitive tools that can be used to correct for a sub-optimal rotation period, it can be shown that they will not work in combination with the clear-cut tax to correct for both harvest percentage and rotation time.

to make sure firms are: (1) cutting down less than 100% of the trees in an area and (2) are actually "thinning" the forest; that they are not simply leaving part of each acre populated and clear-cutting the rest. However, these actions are relatively easy to observe.

Since the firm can choose both the rotation period and the percentage of trees it harvests, and a Pigovian subsidy is unrealistic, a single instrument cannot, in general, control both choices. I analyze the case where the clear-cut tax, combined with the licensing fee can correct the market failure when a logging firm clear-cuts and harvests sooner than socially optimal. I characterize the optimal percentage of trees harvested and rotation time, then analyze how the decisions are affected by the two-part instrument. Finally, I solve for the optimal rates of tax. Furthermore, I analyze the proper application of the tax instrument to forests with different growth and externality characteristics.

3.1 Private Optimization (All Taxes Included)

For convenience of interpretation, I define the following functions:

(2.8) $$b(t) = n(t)g(t)$$

where $n(t)$ is the number of trees, $g(t)$ is the timber volume of a tree at any given time.

[20] The policy maker would have to consider the species of tree being planted. A different number of trees could be planted per acre, depending on the species. Furthermore, different forest types will have a different optimal percentage of uncut trees.

Thus $b(t)$ can be interpreted as the timber volume of the growing trees in the stand at any given time. The timber volume is assumed to be monotonically increasing for all t. Although the number of trees will decrease over time, each tree grows fast enough keep the volume of timber increasing.

To implement the taxes properly, a functional form for the timber value of the forest, $G(T,H)$, must be specified. Let G be broken down into net revenues and costs so that $G(T,H) = pHb(T) - c$, where p is the price of timber net of an per unit harvest costs, and c is a fixed lump-sum harvest and replant cost.[21] The intuition behind specifying c as a fixed cost is that firms must rent a certain amount of equipment and labor to harvest each acre regardless of the harvest percentage; marginal costs, if any, will be captured in p. The term $pHb(T)$ can be interpreted as the value of the growing trees at time T. Now it is possible to define the firm's profit function, net of taxes, at the time of harvest:

$$\pi(T,H) = pHb(T) - c - \tau^{LS} - \tau^{CC}\theta(H) \tag{2.9}$$

The actual penalty incurred from the clear-cut tax is the tax rate, τ^{CC}, multiplied by $\theta(H)$, an increasing, convex function of H. This allows the instrument to take the form of a continuously graduated tax if desired by the planner.

The private landowner chooses T and H to maximize the following value function:

[21] This basic methodology follows Englin and Klan (1990)

(2.10) $$PV(T,H) = \frac{1}{1-e^{-rT}}\pi(T,H)e^{-rT}$$

Notice that the private firm does not internalize the externality. The left-hand side of the equation simply reflects the fact that this private value function depends on T and H.

Maximizing over the rotation period and percentage of trees cut yields the following first order conditions:

(2.11) $$\frac{\partial\pi(T,H)}{\partial T} = \left(\frac{r}{1-e^{-rT}}\right)\pi(T,H)$$

(2.12) $$\frac{d\pi(T,H)}{dH} = 0$$

Equation 2.11 shows that the firm will harvest when the benefit of a marginal increase in T equals the discounted net profit gained by harvesting at time T. Equation 2.12 shows that the firm will choose an H such that the net gains in increasing the harvest percentage are equal to zero. With fixed costs and no taxes the firm will arrive at a corner solution, where $H = 1$.

Plugging the profit function and the proper partial derivates into 2.11 and 2.12 yields:

(2.13) $$pH\frac{db(T)}{dT} = \left(\frac{r}{1-e^{-rT}}\right)(pHb(T) - c - \tau^{LS} - \tau^{CC}\theta(H))$$

(2.14) $$pb(T) = \tau^{CC}\frac{d\theta(H)}{dH}$$

The intuition for 2.13 and 2.14 is similar to that for the previous two equations. The intuition for 2.12 may be somewhat clearer now, as it is apparent that with constant costs and no tax, an internal solution for H does not exist.

3.2 The Growth Function, Externalities, and the Effect of Clear-cutting

Before I launch into the case where the clear-cut tax is used in combination with the lump sum licensing fee, it will be helpful to re-examine the possible forest characteristics that might influence the optimal tax combinations as well as the magnitude of the actual effect once the taxes are implemented.

Before the planner arrives, the private landowner is clear-cutting and harvesting the trees sooner each period than is optimal. I make no prior restrictions on the magnitude of the effects of H and T on the externality, only on the sign. H may have a large effect on the externality in question, as it would be in the case of erosion control on a steep slope, or in the preservation of a species habitat. Or the effect may be a small one, as might be the case if the forest is used for hiking. Furthermore, I make no assumption as to the rate

of growth of the forest. Thus, in the following section I show how the two-part instrument can be used to decrease a firm's harvest percentage and rotation period under fairly general conditions.

3.3 Two Part Instrument: Clear-cut Tax and Lump Sum Licensing Fee

Differentiating 2.13 with respect to τ^{LS} yields the effect of the yield tax on the optimal private rotation period:

$$\frac{dT^*}{d\tau^{LS}} = \left(\frac{r}{1-e^{-rT}}\right) > 0 \tag{2.15}$$

Differentiating 2.13 with respect to τ^{CC} yields the effect of the clear-cut tax on *T**:

$$\frac{dT^*}{d\tau^{CC}} = \left(\frac{r}{1-e^{-rT}}\right)\theta(H) > 0 \tag{2.16}$$

A positive lump sum tax and a clear cut tax will both lengthen the rotation period. This is because both taxes must be paid at the harvest date. A competitive private owner will want to let the trees mature longer in order to reduce the frequency at which the fees must be paid. Since both taxes serve to lengthen the optimal rotation period, the effect of the two-part instrument, where both taxes are positive, is to unambiguously lengthen rotation time. It is also possible that a properly-set subsidy-tax instrument could be used to arrive at the optimal *T*.

Differentiating 2.13 with respect to τ^{LS} yields the effect of the lump sum licensing fee on H^*:

$$\frac{dH^*}{d\tau^Y} = 0 \tag{2.17}$$

Differentiating 2.13 with respect to τ^{CC} yields the effect of the clear-cut tax on H^*:

$$\frac{dH^*}{d\tau^{CC}} = -\frac{d\theta(H)}{dH} < 0 \tag{2.18}$$

The licensing fee has no effect on the optimal harvest percentage. This is expected: the licensing fee penalizes the firm a fixed amount every rotation, but does not penalize it based on how much it harvests. The effect of the clear-cut tax depends upon the marginal penalty received for cutting H. Assuming $\theta(H)$ is increasing in H means the tax decreases the optimal harvest percentage. Further, the net effect of the two-tax instrument on H is negative when both taxes are positive. The policy always decreases the privately-optimal density of cut.

Note that while the clear-cut tax affects both H and T, the licensing fee only affects T. This is both an interesting and useful result. A planner wanting to control for harvest percentage can set τ^{CC} to arrive at the optimal H and then use τ^{LS} to fine tune the

timing of each harvest. If the effect of H on the externality is sufficiently large to produce the need for a large clear-cut tax, inducing an inefficiently long rotation period, τ^{LS} can take the form of a subsidy.

Setting private first–order conditions equal to social first-order conditions, I solve for the optimal tax rates:

(2.19) $$\tau^{CC} = -\int_0^T \frac{\partial F(t,H)}{\partial H} e^{-rt} dt \left(\frac{d\theta(H)}{dH} e^{-rT} \right)^{-1}$$

(2.20) $$\tau^{LS} = \int_0^T (F(t,H) - F(T,H)) e^{-rt} - \tau^{CC} \theta(H)$$

These tax rates make the private first order conditions match the socially optimal first order conditions. Assuming *F(t, H)* decreases in H, the optimal clear cut tax rate always positive. The optimal tax rate increases as the effect of H on the externality increases.[22] For example, if the externality is erosion control, the clear cut tax would necessarily be higher for firms harvesting on sloped landscapes, or above a populated area or water source. Intuitively, the clear-cut tax rate decreases as the tax penalty becomes more convex.

The sign and magnitude of the lump sum licensing fee depends upon the growth rate of the value of the externality and τ^{CC}. The lump sum fee will be positive if:

$$\int_0^T (F(t,H) - F(T,H))e^{-rt} > \tau^{CC}\theta(H) \tag{2.21}$$

or, inserting the optimal τ^{CC}:

$$\int_0^T (F(t,H) - F(T,H))e^{-rt} > -\int_0^T \frac{\partial F(t,H)}{\partial H} e^{-rt} dt \left(\frac{d\theta(H)}{dH} e^{-rT} \right)^{-1} \theta(H) \tag{2.22}$$

If the value of the externality grows slowly throughout the rotation, the licensing fee is likely to be positive and large. Alternatively, if the value of the externality grows rapidly with the age of the forest, as in the case of erosion control, the licensing fee is more likely to be small, or take the form of a subsidy.[23] Furthermore, if the social value of the forest is very sensitive to thinning, requiring a large clear-cut tax, the planner may need to subsidize the firm each rotation. A large τ^{CC} may give the firm the incentive to wait longer to harvest than is optimal. The proper lump sum subsidy would correct the inefficiency.

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[22] Alternatively, if *F(t, H)* is decreasing in *H*, as would be the case forest was otherwise unprofitable to harvest and the externality was fire control, the clear-cut tax takes the form of a subsidy. The subsidy increases as the effect of H on the externality increases.

[23] For example, if erosion control is the externality, the value of *F(t, H)* increases very quickly during the first 10 to 15 years of the rotation, as the trees develop mature root systems, and increases very little thereafter.

In summary, the clear-cut tax affects both harvest percentage per acre and rotation timing. However, the tax alone induces the optimal harvest percentage, but in almost every case causes the firm's rotation period to be too short or too long. Therefore the second part of the two-part instrument, the lump sum licensing fee or subsidy, is needed to correct for the inefficient rotation period.

4. A Simple Numerical Example

So far the model has been very general and has been applicable to a wide range of cases. I now present a more specific numerical model in order to explore in more detail the optimal tax rates under different conditions. This section describes a very simple, but intuitive, stylized example where the clear-cut tax and lump sum tax are implemented.

For the numerical example, I assume a specific form for the for the externality function, *F(H, T)*. The assumed function is shown below and defines the instantaneous value of the amenity for any H and t:

$$F(t,H) = \gamma(1 - H^2)b(\infty) + \gamma H^2 b(t) \tag{2.23}$$

Where γ is the dollar value of the amenity per unit of forest biomass. The first term on the right hand side of the equation can be interpreted as the amenity value of the trees that have been left uncut and have reached peak volume. The second term is the amenity

value of the growing trees that have been replanted since the previous harvest. Squaring the harvest fraction puts more weight on the value of the fully grown trees. This approximates the case where the concern is soil erosion. As the harvested portion of the forest grows back, the new trees will develop root systems and eventually aid in the reduction of erosion. However, since trees are typically harvested long before they are full grown, it is the uncut trees, with fully developed root systems, that will produce the most erosion control.

For simplicity, p is assumed to be net of all costs.

Under these assumptions the new first order conditions for a social optimum become:

(2.24) $$\frac{1}{G(T)}\frac{\partial G(T,H)}{\partial T} = \frac{r}{1-e^{-rT}} + \frac{1}{pb(T)}\frac{\gamma r H}{1-e^{-rt}}\int_0^T (b(t)-b(T))e^{-rt}dt$$

(2.25) $$H = \frac{pe^{-rT}}{2\gamma}\left(\int_0^T \left(\frac{b(\infty)-b(t)}{b(T)}\right)e^{-rt}dt\right)^{-1}$$

The first order condition for the optimal rotation period depends linearly on γ and H, and H depends inversely on γ. Inserting the condition for the optimal harvest percentage into 2.24 shows that the optimal rotation period does not depend on γ.

$$(2.26)\quad \frac{1}{G(T)}\frac{\partial G(T,H)}{\partial T} = \frac{r}{1-e^{-rT}} + \left(1 + \frac{e^{-rT}}{2}\int_0^T (b(\infty) - b(t))e^{-rt}dt\right)^{-1} \int_0^T (b(t) - b(T))e^{-rt}dt$$

For a given H, γ increases the optimal rotation period. However, γ also decreases the optimal H. These effects cancel out exactly and the optimal T is left unaffected by γ. The tax on the harvest fraction will not affect the firm's decision on rotation length.

From 2.25, it is apparent that an internal solution, $0 < H < 1$, will exist if:

$$(2.27)\qquad \frac{pe^{-rT}}{2\gamma} \leq \int_0^T \left(\frac{b(\infty) - b(t)}{b(T)}\right)e^{-rt}dt$$

This is true for a sufficiently small p or sufficiently large γ.

I assume that the clear-cut tax is levied on the square of H: $\theta(H) = H^2$. This penalizes the firm relatively more for a harvest percentage closer to 100%. This reflects the idea that the planner is more concerned with reducing the amount of clear-cutting and cares less if the firm harvests a small fraction of the trees.

The stylized function used for the biomass of trees in board feet, $b(T)$, is based on data

published by Richard MacArdle (1961) for Douglas Fir.[24] The estimated function is:

$$b(T) = \frac{25{,}000}{1 + e^{(-0.2(T-35))}} \tag{2.28}$$

The function is plotted in Figure 2.1. The biomass of this particular stand peaks at 25,000 board feet per acre and reaches this peak at a stand age of 70 years.[25]

The price of a board foot is taken from Calish et. al. (1978) and transformed in the year 2000 dollars. In the model $p =$ \$2.03.

The model is implemented in the Ox programming language (Doornik, 1999). T and H are solved for simultaneously using the conditions above with the estimated parameters inserted. When the real interest rate is assumed to be 3% and γ is assumed equal to \$0.02, the Faustmann privately optimal rotation period (ignoring the externality) is 41.6 years.[26] If the externality is considered, the optimal rotation period is 42.4 years and the optimal percentage of trees harvested per acre is 55%. The optimal clear-cut tax is \$37,444 per square fraction of each acre for a total penalty of \$11,327 per acre. The optimal lump sum payment per rotation period turns out be a subsidy of \$8,891 in this

[24] The MacArdle (1961) data counts the amount of wood available from root to tip of each tree. Although a good estimate of the wood available to a pulp and paper firm, it does not provide an estimate of the wood available to be used as lumber. Thus, using the MacArdle data as a reference, the "stylized" biomass estimation captures roughly the growth in mass of the wood available for lumber.

[25] Obviously, trees will continue to grow past 70 years, but this model assumes that the wood useable for lumber will reach near maximum volume by 70 years.

case. Before taxes, the firm makes a gross profit of $22,822 per acre. The firm makes an after-tax net profit of $20,302 per acre.

Thus, as predicted by the general theoretical model, it is possible that the lump sum licensing fee will take the form of a subsidy in order to correct for the heavy clear-cut tax necessary to reach the optimal harvest percentage. In the case above, the firm is charged a clear-cut penalty that is nearly half of its gross revenue on timber. Furthermore, the firm is harvesting half of the lumber it would harvest in the absence of the tax. Therefore, to reach the optimal rotation time and make up for the clear-cut penalty that induces the optimal harvest percentage but misses the optimal rotation time, the firm must be subsidized each rotation period.

To better illustrate this point, the model can be run under the previous parameter settings but with the tax rates set to incorrect values. For example, the lump sum tax can be set to zero and the clear-cut tax rate held at what was optimal in the previous example ($37,444/acre). Under this tax schedule, the firm will choose a rotation period of 46 years and a harvest percentage of 61%. Thus the firm lets the trees grow for four years longer than is socially optimal, and because of the longer rotation period, the firm harvests 11% more trees than is optimal in order to maximize net profits. Without the corrective lump sum subsidy, the logging firm will harvest too much, too late.

[26] This is a point of reference only. In reality, as in the rest of the analysis, these parameter values will

To evaluate the sensitivity of the model to the assumptions about r and γ, the model was run with a range of values to see how changes in those parameters affect the results. The effects of changing the discount rate, holding γ constant, on the optimal rotation period and harvest percentage are shown in Figure 2.2. The optimal harvest percentage decreases fairly rapidly as the discount rate increases. If the discount rate is high, the present value of the amenities a tree generates (which begin flowing immediately) will outweigh the present value of harvesting the tree at the end of each period--the amenity is worth relatively more when the discount rate is higher. Although T is not affected by the externality, it is affected by the discount rate. It is apparent from the figure that the higher the discount rate is, the shorter the rotation period. If the firm's revenue stream is discounted at a higher rate, it will harvest sooner. The decision to harvest a certain percentage of trees is more sensitive to changes in the discount rate than is the decision on the optimal rotation period. With a discount rate of 7%, as suggested by the Office of Management and Budget (1992), the optimal rotation period is 37.7 years and the optimal harvest percentage is 18%. Thus the optimal rotation period decreased less than five years from the previous example, where r = 3%, but the optimal harvest percentage decreases by 37%.

The effects of changing the marginal amenity value, γ, holding r constant, on the optimal rotation period and harvest percentage are shown in Figure 2.3. As predicted, the optimal rotation period is unaffected by the marginal value of the amenity.

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vary.

Intuitively, the optimal H decreases as γ gets larger. As the value on the x-axis doubles, the optimal H decreases by one half. There is an inverse relationship between harvest percentage and marginal amenity value. At γ = \$2.56/boardfoot, the optimal harvest becomes no harvest at all. This is similar to the case where each tree cut down might drastically increase the chance of a landslide or damaging erosion. And although not shown on the figure, values of γ near \$0.01 produce a corner solution, where it is best to clear-cut. The marginal amenity value of each tree is so low that it is better to chop all of the trees down to reap the timber value.

The different optimal tax rates for changing values of r and γ are shown in Table 2.1. As expected, the clear-cut tax rate increases as the interest rate or gamma increases. However, the actual tax penalty, $\tau^{CC}*H^2$, decreases. This is due to the graduated nature of the tax. Notice that in no case is the lump sum transfer positive. Therefore, in every non-corner-solution case under the assumptions of this particular model, the dual tax instrument will take the form of a clear-cut tax and a lump sum licensing subsidy. It's likely that a different set of assumptions on the functional form of the externality would yield different results.

5. Concluding Remarks

The consideration of harvest percentage introduces a formidable, interesting, and useful economic problem. I've shown how a dual instrument, a clear-cut tax couple with a lump

sum tax or subsidy, can be used to remedy an inefficiency where a valuable forest amenity is underprovided by a firm that is harvesting too many trees too fast. The taxes would be fairly easy to implement and could be used in a wide variety of realistic situations. Allowing each tax to take the form of a penalty or a refund allows for the use of the tax scheme to correct for overgrown forests, therefore aiding in wildfire management, and to correct for rotation periods that may be too long when the forest is being managed for the benefit of wildlife needing a less mature habitat.

The stylized numerical model shows the wide variety of conditions where the clear-cut tax and lump sum transfer could be used to achieve a socially optimal solution when the forest amenity is something like erosion control. In the future, this model could be refined and expanded upon in many ways. It would be interesting to consider different types of amenities. For instance, when wildfire control is considered, it becomes necessary keep the harvest fraction above zero. The externalities function with respect to the harvest fraction may, in this case, be U-shaped. Thus it is possible a harvest percentage subsidy would be optimal to entice a firm to maintain a forest that is unprofitable with respect to timber value.

Chapter III
A Risk Assessment of Bisphenol-A: Accounting for Medical Uncertainty and Data Limitations in Environmental Health Problems

1. Introduction

This paper estimates the damages due to the unregulated use of Bisphenol-A, an endocrine disrupter found in polycarbonate plastics, dental fillings, the linings of aluminum and tin cans, and in contaminated water supplies. Endocrine disrupters, synthetic chemicals that mimic the effects of human hormones, particularly estrogen, can cause fetal deformities, cancer, and negatively effect sperm production. Estimating the possible damages is important to our current government policy, but is also an interesting and formidable economic problem due to the uncertainty arising in toxicology.

Many problems in environmental economics deal with carcinogenic chemicals and the medical uncertainty that comes with them. And though the science is still uncertain, Congress has already passed three laws, the Food Quality Protection Act (1996), the Safe Drinking Water Act (1996), and the Hormone Disruption Research Act (2002), aimed at dealing with endocrine disruptors. On one hand, this legislation is important in safeguarding the public health. On the other hand, as chemical firms are quick to emphasize, these chemicals are very useful, and we should be very careful in passing legislation that regulates or prohibits them.

The difficulty arises in estimating the per capita dose-response function. First, all of the pathways that people can be exposed to Bisphenol-A must be considered in order to estimate per capita dosage properly. Second, research on endocrine disrupters requires clinical trials, where non-human subjects are typically exposed to doses of the chemical that are much higher than humans encounter in the environment. Furthermore, current research on Bisphenol-A shows that it can alter fetal and child development at very small doses even though negative effects are minimal at high doses.

In this study I use a risk assessment approach to provide a framework for estimating the cost of human consumption of chemicals where the dose-response relationship is uncertain. However, I extend current methodology in several significant ways. First, while risk assessment has come a long way towards quantifying uncertainty and variability in models in past five years, current practice in the field still typically relies on the construction of point estimates, or distributions that rely on at least some point estimates. In this model, I estimate a distribution of damages the chemical may cause. The distribution reflects: (1) uncertainty in the varying doses received by different groups in the population, (2) uncertainty and variability in the physiological response to these doses, and (3) uncertainty and variability in the estimates of the economic costs of these responses. Second, I quantify these uncertainties throughout the model. Furthermore, although risk assessment and risk management are typically thought of separately, this model combines risk assessment and benefit-cost analysis in a framework that can easily be used to analyze the implications of different policies, using current

exposure as a baseline. Though steps have already been taken to regulate endocrine disrupters, no such study has been done.

This particular model concentrates on quantifying the health costs of future cancers caused by fetal exposure to BPA *in utero*. In Part 2 of this paper I give a brief overview of BPA and the potential problems associated with it. Part 3 gives a brief overview of the model as a whole. Part 4 discusses, in detail, the procedures used to estimate the effective dose parameters, including the estimation of consumption of goods that contain BPA by the different demographic groups, the amount of BPA leached from each particular good, and the fraction of the BPA consumed which is actually active in the body. Part 5 discusses the estimation of the dose response parameters. Part 6 discusses the costs of the potential outcomes and the range of potential damages.

2. BPA: Exposure Pathways, the Exposed Populations, and Outcomes

Before diving into the mathematical model, the estimation of parameters, and analysis of results, it is important to discuss the nature of Bisphenol-A (BPA). In any thorough risk assessment analysis, there are three major elements of the problem that need to be analyzed. These are exposure pathways, exposed populations, and outcomes. The rest of this section will briefly discuss the environmental estrogen BPA in the context of these three categories.

2.1 Exposure Pathways

How does one become affected by BPA? This is the first question that needs to be answered in order to properly assess the potential damages caused by the chemical. All relevant exposure pathways must be considered to accurately assess the problem.

With a production rate of over 1.6 billion ponds per year, Bisphenol-A is one of the top fifty most-produced chemicals in the U.S. (Vom Saal and Sheehan, 1998). It is currently unregulated and is used as a chemical intermediate for many industrial products, such as polymers, resins, and flame retardants (NRS, 1999). It can be found in polycarbonate plastics such as some Tupperware containers, bottled water, baby bottles, food and drink cans with lacquer coatings, and some dental sealant (Krishnaan *et. al.*, 1993, Brotons *et. al.*, 1995, Olea *et. al.*, 1996, Mountfort *et. al.*, 1997, Howe *et. al.* 1998, Vom Saal *et. al.*, 1998, Wingender, *et. al.*, 1998, Fung *et. al.* 2000). It can also leach into ground water from waste dumps.

BPA is fairly unstable and leaches into food and liquid that comes into contact with it, particularly during heating or autoclaving. Studies have found 0 to 33 micrograms (0 to 6 parts per billion (ppb)) of BPA in food contained in lacquer coated cans and water from polycarbonate containers (Krishnaan *et. al.*, 1993, Brotons *et. al.*, 1995, Mountfort *et. al.*, 1997, Howe *et. al.* 1998, Vom Saal and Sheehan, 1998). This is below the 10 ppb detection limit of U.S. manufacturers of plastics, but above the level (2 ppb) shown to cause relevant estrogenic activity in mice (Vom Saal *et. al.*, 1998).

BPA can enter the body through the skin, but there have been no studies to suggest a relevant reaction is obtained in any way except direct ingestion of the chemical.

2.2 Exposed Populations

Virtually everyone is exposed to a relevant dose of Bisphenol-A every day. This study limits itself to the U.S. population. Thus, anyone in the U.S. who eats or drinks something out of a can, or plastic container, or gets a tooth filled is exposed to BPA.

Demographic groups differ in their vulnerability to BPA, however. Developing fetuses, for instance, are in particular danger from BPA (Bern, 1992, Vom Saal *et. al.*, 1998, Vom Saal and Sheehan, 1998). Reactions to BPA by adults are probably, at worst, transitory (NRC, 1999). Between the fetus and the adult are three relevant age groups: neonates (ages 0-2 years), prepubescent children (ages 2-13 years), and adolescent children (ages 13 – 18 yrs). Unfortunately, for these groups there have been no human studies on the link between exposure to BPA, or any estrogenic substance, and adverse health effects.[27] Since the adverse outcomes due to prenatal exposure are the most studied, and fetuses are widely accepted as the highest risk group, I focus on the effects of BPA due to ingestion by pregnant women in the United States.[28]

2.3 Outcomes

[27] Female neonatal rodents exposed to Diethylstilbestrol (DES) suffered genital tract lesions and malformations. (Bern, et. al., 1987, Medlock, et. al., 1988, Brody and Cunha, 1989). To the best of my knowledge, no BPA studies have been performed postnatally on any species.

[28] For more detail about other potentially at risk demographic groups, see the Appendix, Section C.

As stated before, Bisphenol-A is an endocrine disruptor, or estrogen mimic. It is approximately 100 times weaker (less estrogenic) than estradiol. However, though it binds less frequently to estrogen receptors, it does so in a similar fashion, and therefore causes some of the very same results (Nagel *et. al.*, 1997).

BPA causes the proliferation of MCF-7, human mammary cancer, cells (Krishnaan *et. al.*, 1993, Brotons *et. al.*, 1995, Olea *et. al.*, 1996, Colerangle *et. al.* Nagel *et. al.*, 1997). Although this does not directly lead to the development of cancer, it can cause latent cases to become active. Furthermore, though there is very little data linking BPA to cancer cases--due to it's relative potency only recently being discovered--there is a large literature linking diethylstilbestrol (DES), another synthetic chemical which mimics estrogen, to testicular and cervicovaginal cancer (Herbst *et. al.*, 1971, Scully *et. al*., 1974, Smith *et. al*., 1975, Ziel and Finkle, 1975, Poskanzer and Herbst, 1977, Forsberg, 1979, Gill *et. al.*, 1979, Henderson *et. al.*, 1979, Herbst *et. al.* 1979, McLachlan, 1979, Rustia, 1979, Schottenfield, *et. al*., 1980, Depue *et. al.*, 1983, Newbold and McLachlan, 1982, Leary *et. al*., 1984, Newbold *et. al*., 1985, Brown *et. al*., 1986, Newbold *et. al*., 1987, Bullock *et. al.* 1988, Walker *et. al*., 1988, Bern, 1992, Greco *et. al.*, 1993, Nandi, *et.* al., 1995, Hatch *et. al.*, 1998). Like estradiol, DES is a much stronger estrogen than BPA. However, similar, albeit less in number, outcomes can be expected from the ingestion of the two chemicals (Nagel, *et. al.*, 1997).

This paper focuses on the damages due to extra cases of testicular and cervicovaginal cancer caused by fetal exposure to BPA.[29] Although BPA may cause other adverse health effects, cancer is by far the most costly adverse outcome.[30] Furthermore, for other outcomes, there is either no clear dose-response relationship, or there is no clear cost of the outcome.[31,32]

3. The Model: A Brief Overview

The model is implemented in the Ox programming language (Doornik, 1999). Surveys of medical literature and econometric testing are used to produce estimated distributions of the parameters included in the model. The distribution of total damages is then calculated using a Monte Carlo analysis. The parameter distributions are used as the source of 25,000 draws of the vector of parameters, and each vector is then fed into the mathematical model described below.

The basic model is shown below:

$$(3.1) \quad I_i = \sum_{1}^{j} \Lambda_{ij} X_j$$

[29] For details on other possible adverse outcomes, see Appendix, section D.
[30] For instance, treatment costs for benign prostatic hyperplasia (BPH) range roughly from $2000 to $11,000 in year 2000 dollars (AHCPR, 1994). More than 90% of males experience some form of BPH by the time they are 65 years or older. However, even if a severe form BPH occurs, on average, at 30 years of age in agents exposed to BPA prenatally, and assuming a relatively low discount rate of 3%, the discounted cost of the most expensive treatment would be little more than $1 per case. Therefore, even if the number of cases due to BPA were large, these costs would swamped by the costs due to increased cancer incidence.
[31] For example, fetal exposure to BPA causes permanent prostate enlargement in mice (Nagel, *et. al.*, 1997). However, it is difficult to ascertain from the data when the onset of the problem would occur in humans and how serious the problem would be, given *any* dose.
[32] In female mice, fetal exposure to BPA can cause an earlier onset of puberty (vom Saal and Sheehan, 1998). It would be difficult to nail down, or to even sign, the actual cost of early puberty.

(3.2) $E_i = k_i * rba$

(3.3) $R_{il} = \left(\mathrm{A}_{il} + \mathrm{B}_{il} E_i + \Gamma_{il} \left(E_i\right)^2\right) - \mathrm{A}_{il}$

(3.4) $M_l = \sum_1^i \left(N_i\right) R_{il}$

(3.5) $C_l = e^{-rT_l} [\left(\Phi_l\right) C1_l + (1 - \Phi_l) C2_l]$

(3.6) $D = \sum_1^l \mathrm{M}_l C_l$

Where I_i is the amount BPA ingested by agent i, Λ_{ij} is the amount of good j consumed by agent i, X_j is the amount of BPA that leaches from good j, k_i is the amount of BPA that reaches the bloodstream of agent i,[33] E_i is the estradiol equivalent dose of BPA received by agent i, rba is relative binding affinity (this is how the relative estrogenicity of BPA is measured), R_{il} is the probability that agent i's exposure to BPA will produce a new case of outcome l, A_{il}, B_{il}, and Γ_{il}, are matrices of dose-response parameters, M_l is the number of new cases of outcome l, N_i is the population of group i, C_l is the cost of outcome l, $C1_l$ is the treatment cost of outcome l, $C2_l$ is the treatment cost plus the value of a life, Φ_l is the chance of death due to outcome l, T_l is the latency period of outcome l, and D is the total predicted damages in dollars

The model can be broken down into three basic pieces that will be discussed in detail in the following sections. The first two equations describe the consumption of goods

[33] k_i is meant to capture the possibility that less BPA will cross the placental barrier to reach the fetus than the mother ingests. However, since 100% of the data is based on ingestion by the mother, not the active amount of BPA in the fetal bloodstream, k_i is equal to one in all cases of this model. It is, however, left in the model in case new data surfaces that would cause it to be different than one.

containing BPA, and the active doses received by each agent. Equations 3.3 and 3.4 describe the relative strength of BPA to other estrogens and the response to the doses received by each agent.[34] Equations 3.5 and 3.6 describe the number of new cases of each outcome that should be expected and the monetary cost of these outcomes.

4. Estimating Dosage

Recall equation 3.1 in the model:

$$(3.1) \quad I_i = \sum_{1}^{j} \Lambda_{ij} X_j$$

It states that ingestion of BPA by an agent in population i from goods 1 to j is equal to the consumption of those goods by the agent multiplied by the amount of BPA that leaches from goods 1 to j.

Thus, in estimating the active dose and agent receives, several things must be considered. How much of each product containing BPA does each agent consume? How much Bisphenol-A is does an agent ingest when she consumes a good that has come into contact with the chemical? These issues are discussed in detail below.

4.1 Consumption of Goods Containing BPA

[34] Relative Binding Affinity will be described in greater detail later in the paper, but is essentially the link between studies using other estrogen-mimicking chemicals and the response that BPA would produce.

The goods containing BPA are broken down into seven distinct categories: canned goods, dental sealant, bottled water, Tupperware and plastic ware, canned juice, soft drink cans, and tap water.[35]

The 1996 Consumer Expenditure Survey (Diary Survey) is used to estimate the expenditure by agents on canned food and canned soft drinks.[36] Several methods were attempted in estimating average expenditures for the different age and gender groups. However, the variance of consumption within groups is very larger relative to the variance of consumption between groups. As a result, it is impossible to discern the difference in spending habits between the groups. It is also difficult to infer from the data the amount of each item consumed by each individual within the household. Therefore, estimations for expenditures are performed by calculating the mean expenditure and the variance for each good within single-female households consisting of women of childbearing age (18–40 years old) only. It is likely that women change their consumption habits when living with a roommate or family.[37] However, the differences are either insignificant or impossible to infer from the data. There is a large percentage of women in the survey who report spending zero dollars on canned food and soft dinks and are therefore assumed to consume zero on average. The expenditures by women of

[35] Baby bottles and beer cans also contain BPA, but pregnant mothers are assumed to consume insignificant amounts through these sources.
[36] Data for juice consumption is also included in the survey. However, the average price was unavailable and probably fluctuates widely depending on the type of juice, serving size, and container type. Therefore, a different method, to be discussed later, was used to calculate the consumption of juice.
[37] Women living together in a two person household, on average, spent roughly double the amount on the relevant goods.

childbearing age who spend a non-zero amount on canned food and soft drinks can be seen in Table 3.1.

To translate expenditures into consumption, average 1996 prices for the goods, calculated by the Bureau of Labor and Statistics, are used.[38] Expenditures are divided by the average prices to calculate consumption. Since the Diary Survey reports expenditures over a two-week time period, the results also are converted to daily consumption. The results can be seen in Table 3.1.[39]

The distributions of consumption of these goods, for those households that consume an amount greater than zero on average, are assumed to be log-normal. However, because the population can actually be split into consumers and non-consumers, the draws from the distribution describing the population's average daily consumption are taken in a two step process. First draws are pulled from a uniform distribution ranging from zero to one. If the draw is less than or equal to the fraction of the population estimated to consume zero, consumption is set to zero. Draws from the uniform distribution that are greater than the fraction of the population consuming zero are mapped into the log-normal distribution that describes the variability in consumption patterns of consumers of the good. For example, I estimate that 65% of women between 18 and 40 years old do not consume soft drinks on a daily basis. Therefore draws from a uniform distribution less

[38] Soda is priced per 12 ounces. The canned goods are priced as the average between canned corn and canned tomatoes, per pound.
[39] Fetuses are assumed to consume exactly what their mother consumes. All data gathered shows a response due to fetal exposure to estrogen, based on doses received by the mother.

than or equal to 0.65 are set equal to zero. Draws from the uniform distribution that are greater than 0.65 are mapped into a lognormal distribution with a mean of 0.78 (12 ounce units) and a standard deviation of 0.57.

Data on water consumption is obtained from the International Bottled Water Association's (IBWA) website.[40] From a survey conducted in 2000, IBWA finds that the average American drinks 6.1 8 ounce servings of water per day. They also find that on average, a person drinks 3.8 servings of tap water and 2.3 servings of bottled water. No confidence intervals around these estimates are reported. They do, however, report that some people drink as few as zero servings per day and others drink as many as 12 servings. Therefore, a triangular distribution ranging from zero to 12 (8 ounce servings), with a mode of 6.1 is used to describe the variability in water consumption. Draws representing total average daily water consumption are taken from this distribution.

To calculate tap water consumption, the draws from total water consumption distribution are multiplied by the average fraction that the IBWA estimates to be consumed as tap water: 3.8 tap water servings divided by 6.1 total servings. Likewise, to calculate bottled water consumption, the draws from total water consumption distribution are multiplied by the average fraction that the IBWA estimates to be consumed as bottled water: 2.3 bottled water servings divided by 6.1 total servings. However, not all bottled water will contain BPA. Only water consumed from three or five gallon water tanks (made of

[40] http://www.bottledwater.org/public/BWFactsHome_main.htm (October, 2002)

polycarbonate plastic) will contain BPA. Therefore the bottled water estimate must also be scaled by the percentage consumed from large water tanks. Since there is virtually no data detailed enough to separate five-gallon bottled water consumption from other bottled water consumption, I assume that between 10% and 50% of all bottled water consumed comes from three or five gallon tanks made of polycarbonate plastic.[41]

For canned juice consumption, data is yet again an issue. The 1996 Consumer Expenditure Survey (Diary Survey) is used to estimate that estimate that 77% of women between 18 and 40 years old do not consume juice on a daily basis. However, since the price of juice varies greatly between types of juice and brand, the expenditure data can not be used to estimate average consumption. Instead, estimates of U.S. production from the USDA's *Fruit and Tree Nuts Yearbook Summary* (2002) are used to calculate that the average American drinks 3.2 ounces of juice daily. However, IBWA's 2000 survey of beverage intake finds that the average person drinks 1.1 servings (8 ounces) per day. These values are used as data points to estimate a lognormal distribution of juice consumption. However, since both the survey and the national data include non-juice drinkers, the mean and standard deviation of the distribution must be adjusted accordingly. The final distribution used to model juice consumption by agents that consume more than zero servings everyday is a lognormal with a mean of 3.26 servings per day and a standard deviation of 1.04.

[41] Since bottled water is recently so readily available in other forms (besides the 5 gallon version) 50% is

There is yet another problem: juice comes in many types of containers. Juice can be packaged in plastic (non-polycarbonate), paper or cardboard, glass, or cans. To my knowledge, data breaking the consumption of juice down to this detail is unavailable. Therefore, I assume that between 10% and 50% of juice consumed is consumed from cans. Each draw from the triangular distribution described above is scaled by a draw from a uniform distribution ranging between 0.1 and 0.5. The simulated data will contain both variability and uncertainty in the estimate of juice consumed from cans.

No data could be found concerning the daily use of Tupperware and plastic ware. I assume that the lower bound on average use of Tupperware and plastic ware is 0. I assume the upper bound is not higher than the number of meals an average person will eat in a day. Therefore the upper bound is set at 3. The distribution between these two points is assumed to be uniform. Not all Tupperware and plastic ware is made of potentially BPA-leaching polycarbonate plastic. In fact, very few of the products considered "Tupperware" or plastic ware (including products by Tupperware, Rubbermaid, Playtex, Ziploc, etc.) are made from polycarbonate plastic.[42] However, no actual percentages of the products marketed containing polycarbonate plastic are published. For the purpose of this paper, the percentage of plastic storage items and utensils used that contain BPA is assumed to be between 10% and 40%. Therefore draws from the distribution described above are scaled by draws from another a uniform distribution ranging between 0.1 and 0.4. The data simulated during the Monte Carlo

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likely to be an upper bound.

process should roughly describe the variability and uncertainty involved in the estimation of average use of polycarbonate plastic ware.

The average daily amount of dental sealant procedures is calculated using data from the American Dental Association's "The 1990 Survey of Dental Services Rendered". The number of possible teeth an agent can get sealed varies with the agent's age. In calculating the average amount of sealant each fetus is exposed to, the model multiplies the number of teeth an agent might get sealed by the probability that a particular agent will get the procedure done on any given day, and again by the probability that the agent is pregnant.[43] Needless to say, this number is very small. Furthermore, the sealant does not leach detectable amounts more than three hours after application (Fung *et. al.* 2000), so the exposure is a one-shot dose. However, since a fetus exposed to even very low doses of BPA during gestation may experience an adverse outcome, it remains important to include dental sealant exposure in the model.[44]

If an agent has a sealant procedure done, the variability in the number of number of teeth that an agent has sealed is captured by a triangular distribution ranging between 1 and the

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[42] http://www.chnet.org/healthhouse/pdf/plasticchart.pdf, (October, 2002)

[43] The probability that a fetus is exposed to BPA leached from dental sealant is the probability that a female gets the sealant procedure done on a given day times the probability that she is pregnant. The probability that a woman is pregnant (with a male or female child) on a given day is calculated by dividing the number of births (male or female) in a year by the population of women in the U.S.. The number of births and the populations estimates are taken from the 2000 U.S. Census, conducted by the U.S. Census Bureau.

[44] Though most of the dose response data is taken from studies where subjects were given either daily or weekly doses through much of the gestation period, the dental sealant exposure is left in the model without discount for two primary reasons: 1.) because it is uncertain whether the total dose of BPA or a dose given on any (or a particular) single day is what drives the adverse response; and 2.) because the probability is

maximum number of possible teeth. For an adult female, the maximum number of teeth that could be sealed on a given day is 16.[45] The mode is four teeth.

4.2 Leaching

Much literature has been written recently about the amount of BPA that is actually ingested when a particular good is consumed. However, the subject is fairly controversial. First, the reported amount of BPA leached from a particular good differs greatly depending on the techniques used for extraction and measurement. Second, none of the consumable goods are actually measured for BPA content. For instance, in a typical study, cans containing a food item are emptied, autoclaved, then the water from the autoclaving process is measured for BPA content (Krishnaan *et. al.* 1993, Howe *et. al.* 1998). To first order, this is a fair measurement, since cans are typically packed directly after autoclaving. However, the liquor and food that is actually packaged in the cans is in contact with the partially unpolymerized resin lining much longer than the autoclaved water. Furthermore, water is neutral but the liquor used to store food is typically acidic. Therefore, these measurements are likely to be lower bounds. Brotons *et. al.* (1995) actually does measure the BPA found in the liquors. Therefore, the Brotons estimates are used in the model.[46]

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very small that a pregnant woman will have procedure on a given day, the model results are relatively insensitive to changes in this particular parameter.

[45] This does not include wisdom teeth that are rarely sealed.

[46] See Appendix D for more detail on the Brotons *et. al*. data and how the canned food leaching parameter is calculated.

There are two articles reporting leaching from dental sealant (Olea *et. al.*, 1996, Fung *et. al.* 2000). These also differ in methodology and results. Results from each paper are used to calculate the amount of BPA leached per gram of tooth sealed.[47]

Data for the rest of the BPA leaching from containers is obtained from Takao *et. al.* (1999). Takao reports direct experimental results for the amount of BPA (in or ppb or μg/l) that can be found in groundwater, soft drink cans, and polycarbonate baby bottles. Tupperware and water bottles are assumed to leach BPA at a similar rate to polycarbonate baby bottles.[48]

Canned juice either comes in can similar to those holding food or similar to those holding soft drinks. To calculate the BPA leaching into canned juice, it is assumed similar to the average of the amount that leaches from food cans and the amount that leaches into soft drink cans.

4.3 Dosage Received Results

The results of the Monte Carlo simulation for the I_i distributions can be seen in the Table 3.2 and Chart 3.1. The distributions reflect both uncertainty and variability in consumption habits and variability in leaching. Since the draws for male and female fetal exposure are taken from identical (except in the case of dental sealant) distributions, the

[47] See Appendix D for more detail on the Olea and Fung data and the calculation of dental sealant the leaching parameter.
[48] See Appendix D for more detail on the Takao *et. al.* data and the calculation of the relevant leaching parameters.

statistics for each are not significantly different from each other. More than 50 percent of the average exposure to BPA is through consumption of goods stored or prepared in polycarbonate plastic ware. Roughly 15% comes from canned food and 13% from tap water. As expected, the average daily exposure due to dental sealant accounts for a relatively insignificant percentage (< 0.1%) of the total exposure.

The estimated distribution of average total daily ingestion of BPA by pregnant women is roughly lognormal, has a mean of 8 μg, a standard deviation of 5.4 μg, and a 95% confidence interval ranging between 1 and 21 μg. The maximum value drawn from the distribution is 99 μg. This range is well below the 50 mg/kg dose under which the plastics industry finds no adverse effect in animals tested using high dose methods (vom Saal and Sheehan, 1998). However, vom Saal and Sheehan (1998) assert that there is no dose under which there is no observed effect. Nagel *et. al.*, (1997) show significant effects on offspring of mice fed 2 μg/kg body weight during gestation.[49] This translates roughly to a dose between 100 and 150 μg for a pregnant human. Thus, these potential exposure ranges, based on reasonable average consumption and leaching estimates, are extremely low. This fact will play an important role in the results that follow.

5. Estimating Dose Response

Estimating the dose response function is not as simple as building a good model, getting the data, and running a regression. First, the realization that Bisphenol-A is relevantly

[49] This is the lowest dose study published at the time of this study.

estrogenic and that it is present in many of things we consume is a fairly recent development. So there are no studies of the effects of BPA on human subjects and relatively few on animals. Second, the studies that are conducted vary in procedure and are typically performed using unrealistically high doses. To combat the first problem, I obtained data from experiments with other chemicals with similar properties, such as estradiol and diethylstilbestrol (DES). There have been numerous studies, including ones with human subjects, about the effects of DES. It is a much stronger estrogen mimic than BPA (Nagel *et. al*., 1997), so using the data to infer damages potentially caused by BPA requires an intermediate step. I use a measure of estrogenicity called relative binding affinity (RBA) to tie the DES and estradiol studies to BPA. RBA is discussed in greater detail below. To combat the second problem, care must be taken to understand how procedures may have affected the outcomes of each study. Furthermore, because low dose--or relevant dose--studies are sparse, it is important to correctly account for variance in the estimations.

5.1 Relative Binding Affinity

The relative level of bioactivity of estrogenic substances, or estrogenicity, is typically measured by "relative binding affinity" (RBA). RBA is calculated by measuring how much MFC-7 cell proliferation a certain amount of a chemical causes under controlled conditions.[50] The chemical being tested competes with estradiol to bind to estrogen

[50] MFC-7 cells are human mammory cancer cells.

receptors. A chemical's "affinity" for the estrogen receptors is proportional to how well it competes with the estradiol.

There are many different variations of the test and, of course, many variations in the results each test produces. Not long ago the literature had a difficult time explaining why Bisphenol-A, when tested for relative binding affinity *in vitro* was only weakly estrogenic, but was producing profound effects when tested *in vivo*. Nagel *et al.* (1997) developed a test that seems to explain the discrepancy. When the standard *in vitro* assays are modified with human blood serum, the measured RBA for Bisphenol-A becomes a much better indicator of actual *in vivo* effects. Because the tests developed by Nagel *et. al.* are able to most accurately predict the level of bioactivity Bisphenol-A will produce in mice and rats, I use their numbers for the relative binding affinity parameter, *rba*, in the model.

(3.2) $E_i = k_i * rba$

This measurement allows me to tie DES studies to the BPA model in equation 2 shown above.[51] Nagel *et. al*. show that the relative binding affinity of BPA to DES is 0.01 with a standard error of 0.0012. The distribution describing the variability around Nagel's mean estimate of relative binding affinity is assumed to be normal.

[51] As stated earlier, k is assumed to be equal to one due to the nature of the data collected for this study.

5.2 Dose Response Data

As stated previously, because the relatively high estrogenic strength of BPA has only recently been discovered, there is very little data linking it directly to cases of cancer. [52] However, there is a large literature linking DES, or diethylstilbestrol, to testicular and cervicovaginal cancer (Herbst *et. al.*, 1971, Scully *et. al*., 1974, Smith *et. al*., 1975, Ziel and Finkle, 1975, Poskanzer and Herbst, 1977, Forsberg, 1979, Gill *et. al.*, 1979, Henderson *et. al.*, 1979, Herbst *et. al.* 1979, McLachlan, 1979, Rustia, 1979, Schottenfield, *et. al*., 1980, Depue *et. al.*, 1983, Newbold and McLachlan, 1982, Leary *et. al*., 1984, Newbold *et. al*., 1985, Brown *et. al*., 1986, Newbold *et. al*., 1987, Bullock *et. al.* 1988, Walker *et. al*., 1988, Bern, 1992, Greco *et. al.*, 1993, Nandi, *et.* al., 1995, Hatch *et. al.*, 1998). Many of these studies are "case-control" studies, where a group of cancer patients (case) and people without a history of cancer (control) are questioned to see if they were exposed to various things that may have led to the cancer, including DES. These types of studies are able establish a relationship between cancer and DES exposure, but cannot be used to establish a dose response relationship. To estimate a dose response curve, data that convey what the incidence rate of cancer is among those exposed to DES, and how much they were exposed to, is needed. There are few studies that fit the criteria, and the doses received in the human studies are uncertain. Therefore it is necessary to include data from studies performed on mice. Although not ideal, these studies are useful since the underlying mechanisms of the action of hormones are fundamentally the same across all vertebrates (vom Saal *et. al*., 1998).

[52] Krishnaan *et. al.* (1993) were the first to find that BPA was more estrogenic than previously thought.

To estimate the dose response function for testicular cancer due to DES exposure, I use data obtained from Leary *et. al*. (1984), and Newbold *et. al*. (1985, 1987). The Leary study contains data for 781 DES intrauterine-exposed subjects and 676 unexposed controls. Two of the DES exposed men had a history of testicular cancer. None of the men in the control group had cancer.[53] The exact dose of each study participant is not known, but the average daily dose per quartile during gestation of the population is known (5.5 mg/day, 6.9mg/day, 10.4 mg/day, and 23.3 mg/day, respectively). These mean doses are use to break the exposed data into 4 separate dose groups, each with same testicular cancer incidence rate, and a zero dose group with a zero incidence rate. Both Newbold papers are studies on mice. The 1985 study contains data on 233 pregnant mice exposed to 100 μg/kg of body weight daily during gestation and 96 unexposed controls. The incidence rate of testicular cancer among offspring of the exposed group was 4.7% and zero among the controls. The 1987 study provides data on 277 pregnant mice exposed to 100 μg/kg of body weight daily during gestation and 122 controls. Offspring of the exposed group had a testicular cancer incidence rate of 1.8%. The incidence rate of controls was zero. All doses used in the data set are converted to micrograms and, in the mouse studies, adjusted to reflect a similar dose for a human weighing 70 kg.[54]

[53] The cancer incidence in both groups may be underestimated, since the ages of those in the study were between 18 and 45 years.

[54] In addition to a correction for weight, a correction for free estradiol in the serum of the agent should be used in future versions of the model. The more free estradiol present in the system, the less effective BPA will be. Humans typically have a greater ratio of free estradiol than rats, therefore this data may overstate

To estimate the dose response function for cervicovaginal cancer due to DES exposure, I use data obtained from Newbold and McLachlan (1982) and Hatch *et. al*. (1998). The Hatch study examines the cancer rate among a total of 4536 intrauterine DES-exposed women and 1544 unexposed controls. The cervicovaginal cancer incidence rate among the exposed women was 0.066%. The incidence rate among the controls was zero.[55] Since exact doses for the women exposed prenatally is not known, the dose data from Leary *et. al*. (1984) is used to approximate the doses for quartiles of the sample, as explained in the previous paragraph. In the Newbold and McLachlan study, data for 91 mice exposed prenatally exposed to various doses of DES and 158 unexposed controls are reported. Of 29 mice exposed to 5 μg/kg/day of gestation, one contracted an adenocarcinoma in the vagina. Zero of 16 mice exposed to 10 μg/kg/day contracted cancer. One of 46 mice given 100 μg/kg/day prenatally got cancer. The incidence rate among controls was zero. All doses used in the data set are converted to micrograms and, in the mouse studies, adjusted to reflect a similar dose for a human weighing 70 kg.

Note that *all* of the data described above should be considered high dose data, since the estimated daily amount of BPA exposure adjusted for RBA is equal to somewhere between 0.01 and 0.21 μg (with 95% confidence) of daily DES exposure.

5.3 Estimating the Dose Response Function

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the effects of BPA in humans. The current problem is that a reasonable estimate of the free estradiol ratio in human fetal serum is unavailable.

Typical risk assessment studies use a linear or exponential function to estimate dose response (Griffiths, *et. al.*, 2002). To deal with low dose (dose ranges below their lowest observed dose) estimation, they force the function through the origin by drawing a line, or a curve, from the response level at the lowest observed dose through the origin, or some value along the x-axis deemed to be the threshold dose (Griffiths, *et. al.*, 2002). This method has two major problems: (1) it assumes monotonicity and (2) it does not properly account for the natural incidence rate of the outcome or the variance around the low doses.

In all vertebrates, including humans, an active amount of free estradiol is already present in the body. Therefore, according vom Saal *et. al.* (1998), vom Saal and Sheehan (1998) and Nagel *et. al.* (1997), *any* perturbation by the ingestion of estrogen-like substances will cause a significant reaction. Furthermore, as the exposure levels increase and estrogen receptors in the system become saturated, the response to a marginal increase in an estrogen mimic may decrease. Therefore, there is no threshold dose (dose below which there is no observable reaction) and the response function may be nonmonotonic.

Using the data described above, a quadratic function, shown below, that allows for nonmonotonicity in predicting the response to a DES equivalent dose of BPA, is estimated. Note that from here on, for stylistic simplicity, I refer to exposure to a "DES equivalent dose of BPA" as simply as "a dose of BPA".

[55] Again, the cancer incidence in both groups may be underestimated, since the ages of those in the study

$$(3.3) \quad \underbrace{R_{il} = \left(\mathrm{A}_{il} + \mathrm{B}_{il} E_i + \Gamma_{il} \left(E_i\right)^2\right)}_{EstimatedFunction} - \mathrm{A}_{il}$$

A_{il} captures the natural occurring incidence rate of outcome l, in agent i, given no exposure to BPA. B_{il} and Γ_{il} capture the slope and curvature of the function that describes additional response due to BPA exposure. Thus, this function allows for the removal of the estimated natural incidence rate in the sample in order to calculate the additional number of cancers due only to BPA exposure. The results of the regressions are shown in Table 3.3.[56]

In both response-function estimates for testicular and cervicovaginal cancer respectively, except for the testicular cancer constant term, all estimated coefficients are significant. In both estimations, the F-statistic is significant but the R^2-statistic is extremely low (though the statistics for the testicular cancer regression are significantly higher). The significant F-statistics are encouraging, and the low R^2-statistics are to be expected with so few data points available. The errors around the mean estimates are assumed to be normal.[57]

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were between 18 and 45 years.

[56] Although there were some zero dose response data points, the sample is too small to infer that the incident rate for the population would be zero given a zero dose.

[57] With such a low R^2 statistic, this assumption of normality in the errors may not be a good one. Because very little of the variance is explained by the model, it may be better to use a uniform distribution for the error terms and various confidence intervals around the mean. This may be taken into account in a later version of the model.

Looking at both the estimation results and the estimated functions, shown in Charts 3.2, 3.3, 3.4, and 3.5, it is apparent that both dose response functions are increasing over the relevant range of doses, but nonmonotonic.[58] Although the squared terms in both functions are negative and significant, the estimated coefficients are so small that the function appears to be nearly linear throughout the observed range. Also, for any given dose, the testicular cancer response is greater than the cervicovaginal cancer response by more than a factor of 50. This is also apparent in simulated response distributions shown in Charts 3.6 and 3.7. One possible explanation for this is that cervicovaginal cancer is simply harder to get. Testicular cancer is fairly rare, with an incidence rate of 14 per 100,000 men (SEER database, 2000).[59] However, the adenocarcinomas found in women exposed to DES almost never occur in women not exposed to DES (Herbst *et. al.*, 1979). Another possible explanation is that perturbations of the natural levels of estrogen during gestation may simply affect male offspring more than female offspring.

Also worth noting is that due to (1) the availability of only relatively high-dose study data and (2) the use of DES data as a proxy, the range of environmentally relevant doses observed is very near the origin in Charts 3.2 and 3.4. Therefore in Charts 3.3.and 3.5, the dose response curves are shown only for the environmentally relevant dose range. On observation of these charts, it becomes obvious that the large amount of variance that occurs at high doses nearly disappears completely at such low doses. Even over a total

[58] One drawback of the quadratic function is that, if the coefficient on the squared term is negative and significant, the function will eventually pass through the x-axis. This is counter intuitive. However, in this case the function passes through the x-axis far beyond the relevant dose range. In fact, the function is nearly linear through the relevant dose range.

population exposed of about 4 million, this amount of variance will contribute little to the variance in the final distribution of damages, and the low dose uncertainty is probably underestimated. Though far from perfect, this method is still an improvement over methods that do not directly account for background incidence rates and the possibility of nonmonotonicity in the dose response function.

5.4 Increased Cancer Incidence Results

The number of new future cases of cancer contracted each year, given estimated average ingestion of BPA, is calculated as in equation 3.4:

(3.4) $$M_l = \sum_1^i (N_i) R_{il}$$

The number of new cases of outcome l, M_l, is calculated by multiplying the dose response function by the population.[60] The estimated constant term and the variance around it are subtracted from the response function. Recall the constant term explains the natural incidence rate expected in the sample. Therefore, it must be removed in order to calculate the number of cases attributable only to BPA exposure. The results of the simulation can be seen in Charts 3.8 and 3.9.

[59] This is rate is for men ages 30 to 40, where the incidence rate peaks.
[60] The population of male (female) fetuses in a given year is calculated by multiplying the number of births in 1998 by the ratio born male(female) (2000 census). It is calculated that there are 2,014,287 male fetuses per year and 1,923,713 female fetuses. The population parameters are assumed uniformly distributed between -500 and +500 of the calculated values to account for rounding errors in the census data.

The distribution of new future testicular cancer cases per year caused by BPA exposure has a mean of 0.57 and a standard deviation of 0.39 (with a 95% confidence interval bound between 0.11 and 1.31). This translates roughly to a mean of 0.028 new cases of testicular cancer per year per 100,000 exposed *in utero*, or one new case of testicular cancer due to BPA exposure every other year.

The distribution of new future cervicovaginal cancer cases per year caused by BPA exposure has a mean of 0.01 and a standard deviation of 0.01 (with a 95% confidence interval bound between 0 and 0.03). This translates roughly to a mean of 0.0005 new cases of cervicovaginal cancer per year per 100,000 exposed *in utero*, or one new case of cervicovaginal cancer due to BPA exposure every *100 years*!

As is obvious, the estimated frequency of future cancers stemming from current exposure levels of BPA is extremely low. This is most likely due to very low levels of average ingestion of BPA. However, it is possible, as explained earlier, that the uncertainty in dose response at such low levels of exposure are underestimated in this model. Therefore, sensitivity analysis, discussed in Section 7, is essential in order to understand the robustness of this result.

6. Estimating Damages

The distributions of the costs of each outcome are estimated using equation 3.5 in the model:

$$(3.5) \quad C_l = e^{-rT_l}\left[\left((\Phi_l)C1_l + (1-\Phi_l)C2_l\right)/r\right]$$

C_l is the total cost of an outcome. $C1_l$ is the cost of the outcome, given the agent lives. In this version of the model, it assumed to be equal to the estimated average treatment costs of the outcome. $C2_l$ is the cost of the outcome if the agent dies. It is assumed to be the treatment cost plus the estimated value of a statistical life.[61] Φ_l is the probability, given the agent has the disease, that the agent survives. The costs are discounted over T_l, the estimated latency period of the particular outcome. The total costs are discounted over the full latency period, from birth to the time the disease is discovered. Any benefits of regulation on BPA will not be accrued until the age at which a people would, with the current level of BPA, begin seeing symptoms of the adverse outcomes.

The mean treatment cost in 1990 for cervicovaginal cancer, $44,444, and the standard deviation of the treatment cost is obtained from cervical cancer cost data supplied by the U.S. National Institutes of Health, Office of Science Policy and Planning. The standard deviation for the mean treatment cost is assumed to be similar to that calculated for ovarian cancer in Fireman *et. al.* (1997).[62] The estimated treatment cost of ovarian cancer had the highest estimated standard error, relative to other cancers studied in

[61] The estimated costs do not include other indirect costs, such as pain and suffering or lost work days. Furthermore, the estimates do not account for the severity of the outcome, except with the survival rate. Later versions of the model should include this detail.

[62] Fireman *et. al.* (1997) provides a comprehensive estimate of treatment costs of various types of cancers. Unfortunately, neither cervicovaginal nor testicular cancers are represented in the study. The NIH data contained mean estimates of cost, but no estimation of variance.

Fireman, so this should provide an upper bound on the variability around the estimates for the treatment of cervicovaginal cancer. The errors are assumed to be normally distributed.

The range of possible treatment costs in 1992 for testicular cancer, $29,300 to $47,360 was obtained from the Testicular Cancer Resource Center (2003). All treatment cost estimates are converted to year 2000 dollars using the GDP deflator published in the 2002 Economic Report of the President.

The distribution of the value of a statistical life (VSL) is calculated using data obtained from the EPA's, "The Benefits and Costs of the Clean Air Act 1990 to 2002" (1999). The EPA provides a list of estimates of 26 policy-relevant value-of-life studies. The Landefeld (1979) survey method used to determine the value of avoiding death due to cancer is added to the list because of its particular relevance to this study. The full list used is shown in Table 3.4. The value of a statistical life is difficult to estimate and the uncertainty as to the "correct" VSL must be portrayed with a distribution. Therefore, a log-normal distribution, with a mean of $5,708,000 and a standard deviation of $4,040,000 (in year 2000 dollars), is estimated using the data provided.

Survival rates can be expected to vary due to the progress of the disease before diagnosis and due to individual characteristics. The survival rate of those with cervicovaginal cancer (62.5% - 66.0%) is assumed to range uniformly between the survival rates for

cervical cancer in the years 1990 and 1999 as calculated by the NIH (2003), Office of Science Policy and Planning. The 93.3% mean survival rate for testicular cancer is found using the SEER data base. A 10% range around the mean is assumed.[63] Therefore, a uniform distribution ranging from 88.3% to 98.3% is used in the model.

The results of the simulation for undiscounted costs per additional case of cancer are shown in Charts 3.10 and 3.11.

Due to higher direct costs and a lower survival rate, cervicovaginal cancer is, on average, nearly five times more costly per case to treat than testicular cancer. The model results show that cervicovaginal cancer has a mean cost of $2,147,492 and 95% confidence interval ranging from $1,447,448 to $4,497,910. Testicular cancer has a mean cost of $433,677 and 95% confidence interval ranging from 139,151 to 968,004. The wide confidence intervals can be attributed mainly to uncertainty in the value of statistical life.

The distribution for the latency periods – from birth to the onset of symptoms – for testicular and cervicovaginal cancer are estimated using data from the SEER database. SEER publishes incidence rates for different cancer sites for five-year age groups. This data is used to produce a sort of "step" function, where different age groups have a different odds of being drawn on a given Monte Carlo run, but ages within an age group have an equal chance of being drawn any on any given run. Thus, multiple uniform

[63] In future versions of the model, the mean survival rates, as estimated in the SEER database, should be

distributions, each covering a range of five years, are connected to form the final distribution and account for variability in the latency periods. Draws from the distribution produced a mean latency of 34 years and 55 years for testicular and cervicovaginal cancer respectively.

The Office of Management and Budget (1992) suggests that U.S. government agencies use a real interest rate of 7% in cost-benefit calculations. However, the discount rate is fairly controversial, and no one really knows what the "correct" discount rate is. Thus it is necessary to capture some uncertainty in the OMB estimate. To correctly capture all of the variability and uncertainty throughout the entire model, the uncertainty as to the "correct" discount rate is first portrayed with a distribution. In the model, the uncertainty in the discount rate is captured with a triangular distribution ranging between 3% and 9%, and with a mode of 7%.

The total damages (in year 2000 dollars) caused by unregulated use of BPA are calculated in equation (3.6):

(3.6) $$D = \sum_{1}^{l} \mathrm{M}_l C_l$$

Total damages equal the sum of the new cases of cancer attributable to BPA exposure multiplied by the cost of each case. The estimated damage statistics are shown in Table

used and the number of cancer survivors should be drawn from a binomial distribution.

3.6. The estimated distributions of total and annual damages are shown in Chart 3.12 and Chart 3.13 respectively.

Due to the small number of additional cancers caused by BPA, and the long latency period, the mean estimated damage due to BPA exposure is a relatively small $332,377 even though the population exposed is large and the adverse effects are serious. The undiscounted mean annual costs are estimated to be $271,791 per year. As expected, because the additional frequency of cervicovaginal cancer to BPA is so small, and the latency period is very long, the damages due to this type of cancer contribute only 21.3% to the total estimated damages. Roughly 78.7% of the total damages are due to additional cases of testicular cancer.

The distributions are wide for both the estimated annual and total damages with 95% confidence intervals ranging from $43,808 to $748,139 per year and $11,347 to $1,304,023 total. Furthermore, the Chart 3.12 shows that the mode of the distribution of total damages is close to zero, whereas the mode for annual damages is around $100,000 per year. This information, along with the fact that the distribution of estimated total damages is significantly wider than the distribution of estimated annual damages means that the model results are sensitive to the discount rate. Furthermore, since there is very little variance in the dose-response function near the origin, this uncertainty in the annual and total damages must also stem from the uncertainty in the value of a statistical life

estimate and the variation in the consumption of goods containing BPA. The following section analyzes the model's sensitivity to these uncertainties more thoroughly.

7. Model Analysis

As discussed briefly in the previous section, the uncertainty in the final damage distribution comes from a few different sources with in the model. Reducing the variance or uncertainty in different variables will certainly reduce the amount of uncertainty in the final estimate. However, the most interesting story in this particular model turns out to be how low the mean estimated damages are. Is it the actual data, or an assumption about a parameter or the structure of the model that drives this result? This is the key question examined in this section. Therefore the sensitivity analysis below emphasizes how certain parameters shift the mean.

Charts 3.14 and 3.15 show the new estimated distributions of total damages when the discount rate is set to 7% and 3% respectively. The real discount rate of 7% is chosen both because it is relatively high and is suggested for use in benefits-costs analysis by the OMB (1992). Although 7% is just one percentage point higher than the mean of the uniform distribution used in the base model, the change produces a nearly 50% decrease in mean total damages. This reduction from $332,377 to $178,977 is indeed significant. Thus, it is expected that lowering the discount rate to 3%, a rate that is still realistic but closer to the zero discount rate desired by some environmental groups, would drastically increase the mean estimated damages. When the 3% discount rate is implemented in the

model, the result is a near 500% increase in the total damages, from $332,377 to $1,991,183 with a 95% confidence interval ranging from $259,177 to $6,022,751. The model results are certainly sensitive to the discount rate, which in retrospect shows the importance of including a distribution rather than a point in the original model. However, even reducing the rate to 3% does not produce damage estimates that are likely to be significant compared to the potential costs to the plastics industry and the U.S. economy were BPA to be regulated.

Charts 3.16 and 3.17 show the new estimated distributions of total estimated damages when the value of a statistical life (VSL) is set to roughly double the mean of the distribution used in the original version of the model and the mean, respectively. As expected in a fairly linear model, reducing a distribution to the mean hardly changes the final estimate of mean total damages. In fact, setting the VSL to $5,788,000 results in a total damage estimate of $330,509, 99% of the base mean estimate. However, nailing the VSL down to the mean decreases the total variance in the estimated distribution of damages by 44%. The value of life measure thus accounts for a significant amount of the uncertainty in the final distribution and, therefore, it is important that the uncertainty of the VSL estimate was included in the original model. When the VSL parameter is increased to $12,500,000, the mean total damage estimated increases by a little more than double the original estimate. Even pushing the VSL estimate up to the 95th percentile of the estimated distribution does little more than double the relatively small mean estimated damages.

Thus the uncertainty in the estimates of economic parameters contribute to the uncertainty in the damage estimate but do not explain the very low estimate of total damages due to BPA. As stated earlier, the amount of BPA ingested on average is so low that very few cancers can realistically be attributed to this exposure, even during the critical phases of fetal development. It is possible however, that for some reason or another, the estimated distribution of BPA ingestion understates the amount people are exposed to every day. To examine the sensitivity of the results to ingestion, the model was rerun imposing the assumption that everyone, everyday, ingests what the 99th percentile of the estimated distribution ingest: 25μg. The new distribution of total damages is shown in Chart 3.18. The distribution has a mean of $1,049,056 and 95% confidence interval ranging from $65,714 to $3,840,583. These damage estimates stem from an increase in the mean testicular cancer frequency (over the entire population) from 0.57 new cases per year to year to 1.77 new cases per year and an increase in the cervicovaginal caner frequency from 0.01 new cases per year to 0.04 new cases per year. Though it increases cancer frequencies significantly, even this unrealistic assumption on BPA exposure does not increase the total estimated damages to much more than 1 million dollars.

An alternative approach to sensitivity analysis would be to ask: What piece of the model, if any, could be manipulated to produce a significant amount of total damages, say $100 million or even just $10 million? The dose response function is a key possibility. The

small amount of uncertainty in the dose response function at the relevant but very low dose range is certainly understated. The small range is due to the clustered high-dose data and the assumption of a quadratic does-response function. These assumptions may not be accurate given that very little is known about the response to very small doses of BPA.[64] In the base model, the standard deviation of the slope estimate for both types of cancer is less than 1% of the estimated mean. Thus multiplying the slope by 100, although likely to be outside even the most conservative estimate of dose response, provides for a revision to the model that would sharply increase the estimated damages. The results of this experiment are shown in Chart 3.19. Due to the nearly linear nature of the estimated dose response function, multiplying the slope parameter by 100 results in 100 times the cancer frequency estimated in the primary model. Therefore, under the new dose response function, there would be an estimated mean of 56 testicular cancers due to BPA each year and slightly more than one new cervicovaginal cancer per year. This would result in a total damage distribution with a mean of $33,200,000, and a 95% confidence interval ranging from $1,163,598 to $129,700,000.[65] Though providing a wide range of large damages, scaling the dose-response function up by a factor of 100 still produces damage estimates that are relatively small compared to other substances regulated by the federal government.

8. Concluding Remarks

[64] In future versions of the model, it may be fruitful to estimate different, logical functional forms and make draws from the family of estimated curves.

[65] The uncertainties in the discount rate and the value of a statistical life become much more important in the range of the distribution when the number of cancer cases increase 100-fold.

This paper provides a comprehensive cost analysis, using risk assessment methodology, of the increased risk of testicular and cervicovaginal cancer to those exposed prenatally to current environmental levels Bisphenol-A. The model used to calculate damages tracks variance throughout the model, including in the exposure parameters, the dose-response parameters, and the economic cost parameters. Although uncertainty is a big issue in cutting edge risk assessment, no study to date has tracked variance and uncertainty from consumption to cost. Indeed, using risk assessment methodology to calculate the economic costs of health risks is a fairly new technique.

The resulting estimated damage distribution is broad, with a mean of $332,377 and a 95% confidence interval ranging from $11,347 to $1,304,023. However, even if the true damages lie towards the upper tail, the benefits of BPA to the plastics industry and to consumers is likely to outweigh the health costs incurred by additional cases of testicular and cervicovaginal cancer. There are two reasons for the relatively small amount of estimated damages. First, people are exposed, on average to very small amounts of BPA every day. So, based on the data available and the assumptions in the model, even for the most vulnerable population – fetuses -- there is very little risk of contracting a future case of cancer simply due to BPA exposure. Even results produced with unrealistically conservative exposure estimates (that is, assuming exposure to an unrealistically large dose) resulted in very little additional risk of cancer due to BPA. Second, the decreased health costs due to any regulation will not begin to be realized until nearly 35 years after

the regulation is put into place. Thus any costs of current exposure are discounted to far in the future.

The model illustrates the overall importance of tracking the uncertainty of every parameter in the model. It turns out the damage estimate is very sensitive to the assumptions made on the value of a statistical life and the discount rate. The lack of variance in the dose-response estimation is of some concern. The fact remains that very little is known about the effect of BPA at low doses (except that there probably is one) and the model does not accurately capture this uncertainty. With highly clustered, high dose studies as the only data, most of the variance in the model is explained by the constant variable, the variable explaining the natural incidence rate. Removing the natural incidence rate mean and the variance around it correctly estimates the affects of BPA, but leaves very little variance for doses near zero.[66] As new, lower dose data become available, this problem will become less severe. Until then, a future version of this model should include dose response estimates for a variety of functional forms. This would recapture some of the uncertainty lost when a single function is estimated.

In the future, as data becomes available, besides refining the assumptions currently made on the estimated distributions and parameters, it would be interesting to add in a broader range of populations exposed to BPA, although they may be affected less severely than prenatal children. Furthermore, although it would be unlikely to change the results

drastically, adding in the other non-fatal outcomes, some of which are precancerous conditions, would add complexity and rigor to the model that would be interesting from both a policy and a modeling point of view.

[66] Drawing a line or curve from the lowest observed dose through zero, as is typical in risk assessment, creates the same problem without correctly accounting for the natural incidence rate.

Conclusion

The results in Chapter I show clearly that productive investment is not crowded out by investment in pollution abatement capital in all industries. For the lumber, chemicals, and primary metals industries in fact, it is safe to say that pollution abatement investment actually stimulates productive investment. This result suggests economies of scope for investing in the two types of capital at the same time. Furthermore, the results give empirical support to the Porter's hypothesis and observation (1995) that firms tend to renovate when faced with environmental regulation.

The results are even stronger in light of the assumption that λ is unaffected by investment in abatement equipment. General equilibrium effects cause both interest rates and the price of capital goods to rise when firms demand abatement capital (Wilcoxen, 1988). These effects unambiguously lead to a reduction in productive investment. Therefore, by ignoring them, the observed results are actually biased away from showing a positive correlation between abatement and productive investment.

One possible explanation of this surprising result is the following: If installing pollution abatement equipment requires a section of the plant to be shut down for a period of time, the firm may be able to replace nearby equipment at relatively little cost.

Some caveats must be kept in mind. With data aggregated to the 2-digit level, it is

impossible to see exactly what is happening at the plant or firm level. Regional factors also cannot be observed. This makes it difficult to say exactly what the mechanism is that drives the correlation. It is possible that industries with a few dirty plants located in heavily regulated areas, may have closed those plants and opened new plants in different locations. This would cause a large, positive correlation between abatement and productive investment at the industry level. That phenomenon would consistent with basic observation that abatement investment may stimulate productive investment but the mechanism would not be economies of scope in investment.

Despite the caveats, the results remain very interesting. At the industry level, abatement policy does not seem to crowd out otherwise productive investment in all industries. On the contrary, abatement investment seems to provide an incentive in some industries (via economies of scope) for firms to renovate sooner, and in greater quantities than they normally would. (This does not necessarily imply that individual firms are made better off by regulation, just that in aggregate, their investment rises.) Furthermore, the results show that care must be taken when designing policy affecting a large, aggregate branch of the economy. Since industries react to mandated abatement investment differently, the effect on investment in the productive capital stock of such regulation should be calculated separately for each industry.

These results have implications beyond the range of this paper. Finding economies of scope between other investment pairs will have strong implications for policies designed

to alter the composition of total investment. In fact, it would be surprising if other types of investment did not interact in the adjustment cost function.

In Chapter II, the consideration of harvest percentage introduces a formidable, interesting, and useful economic problem. I've shown how a two instrument, a clear-cut tax couple with a lump sum tax or subsidy can be used to remedy an inefficiency where valuable forest amenity is underprovided by a firm that is harvesting too many trees too fast. The taxes would be fairly easy to implement and could used in a wide variety of realistic situations. Allowing each tax to take the form of a penalty or a refund allows will even allow for the use of the tax scheme to correct for overgrown forests therefore aiding in wild fire management, and to correct for rotation periods that may be too long when the forest is being managed for the benefit of wildlife needing a less mature habitat.

The stylized numerical model shows the wide variety of conditions where the clear-cut tax and lump sum transfer could be used to achieve a socially optimal solution when the forest amenity is something like erosion control. In the future, this model could be refined and expanded upon in many ways. It would be interesting to consider different types of amenities. For instance, when wild fire control is considered it becomes necessary keep the harvest fraction above zero. The externalities function with respect to the harvest fraction may, in this case, be U-shaped. Thus it is possible a type harvest percentage subsidy would be optimal to entice a firm to maintain a forest that is unprofitable with respect to timber value.

Chapter III provides a comprehensive cost analysis, using risk assessment methodology, of the increased risk of testicular and cervicovaginal cancer to those exposed prenatally to current environmental levels Bisphenol-A. The model used to calculate damages tracks variance throughout the model, including in the exposure parameters, the dose response parameters, and the economic cost parameters. Although uncertainty is a big issue in cutting edge risk assessment, no study to date has tracked variance and uncertainty from consumption to cost. Indeed, using risk assessment methodology to calculate the economic costs of health risks is a fairly new technique.

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realized until nearly 35 years after the regulation is put into place. Thus any costs of current exposure are discounted to far in the future.

The model illustrates the overall importance of tracking the uncertainty of every parameter in the model. It turns out the damage estimate is very sensitive to the assumptions made on the value of a statistical life and the discount rate. The lack of variance in the dose-response estimation is of some concern. The fact remains that very little is known about the effect of BPA at low doses (except that there probably is one) and the model does not accurately capture this uncertainty. With highly clustered, high dose studies as the only data, most of the variance in the model is explained by the constant variable, the variable explaining the natural incidence rate. Removing the natural incidence rate mean and the variance around it correctly estimates the affects of BPA, but leaves very little variance for doses near zero. As new, lower dose data become available, this problem will become less severe. Until then, a future version of this model should include dose response estimates for a variety of functional forms. This would recapture some of the uncertainty lost when a single function is estimated.

In the future, as data becomes available, besides refining the assumptions currently made on the estimated distributions and parameters, it would be interesting to add in a broader range of populations exposed to BPA, although they may be affected less severely than prenatal children. Furthermore, although it would be unlikely to change the results drastically, adding in the other non-fatal outcomes, some of which are precancerous

conditions, would add information, complexity and rigor to the model that would be interesting from both a policy and a modeling point of view.

Tables and Figures

Table 1.1: Manufacturing Industries

SIC	Description	SIC	Description
20	Food	30	Rubber and plastic products
22	Textile mill products	32	Stone, clay, and glass products
24	Lumber and wood products	33	Primary Metals
25	Furniture and fixtures	34	Fabricated metal products
26	Paper and allied products	35	Machinery, except electrical
27	Printing and publishing	36	Electrical machinery
28	Chemicals and allied products	37	Transportation Equipment
29	Petroleum refining	38	Instruments

Figure 1.1: Correlation Between Productive and Abatement Investment (1973-1993)

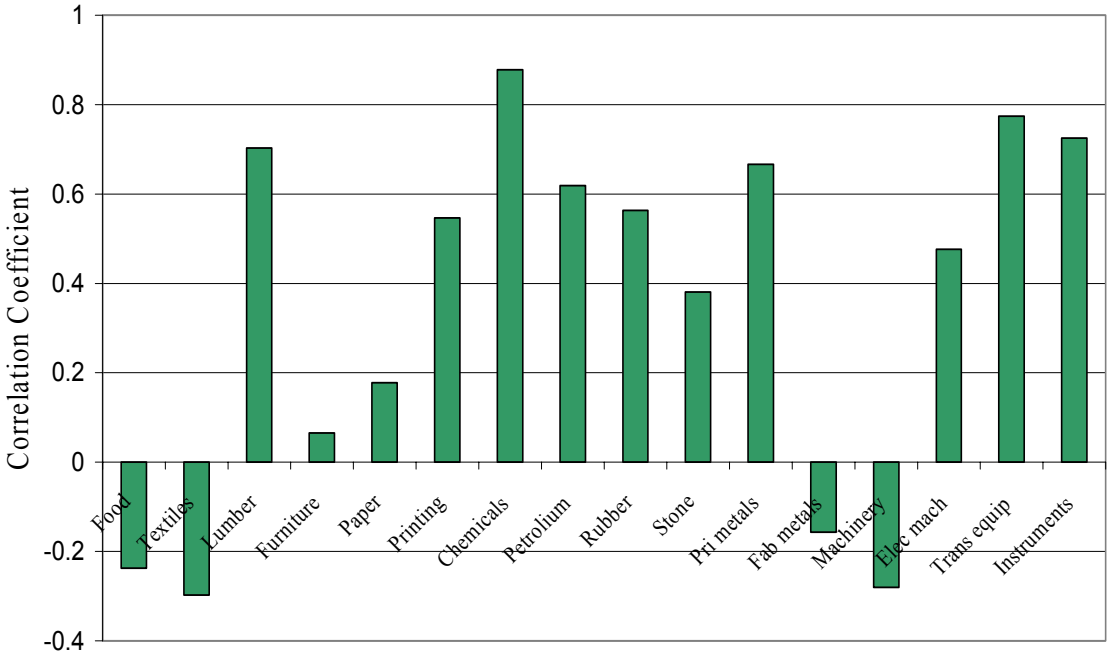


Figure 1.2: Abatement Investment as Percentage of Total Investment, by Industry

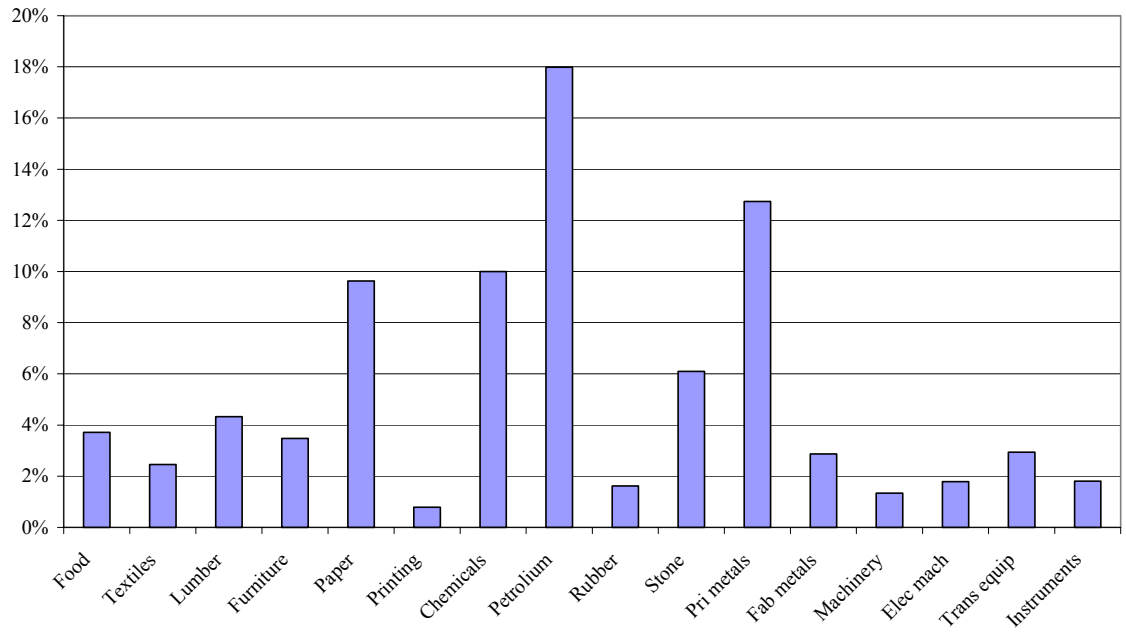


Figure 1.3: Variances (in millions) in Measured Productive and Abatement Investment by Industry

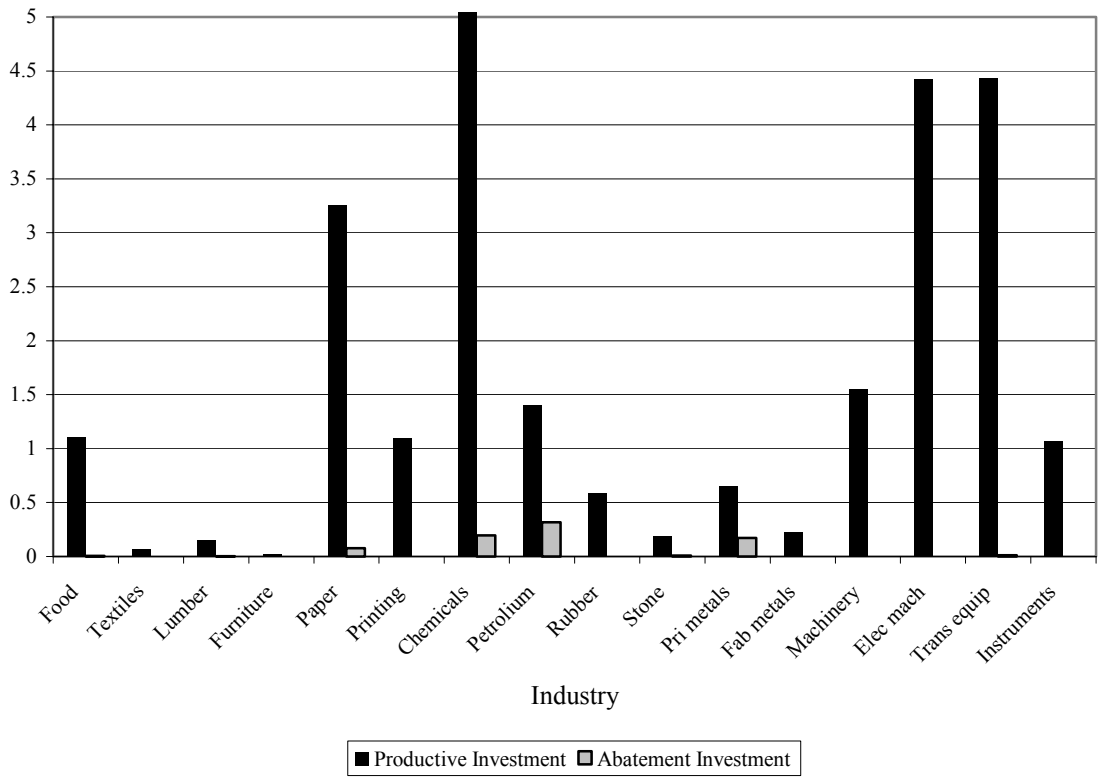


Figure 1.4: % Change in Productive and Abatement Investment for All Industries Between 1973 and 1993

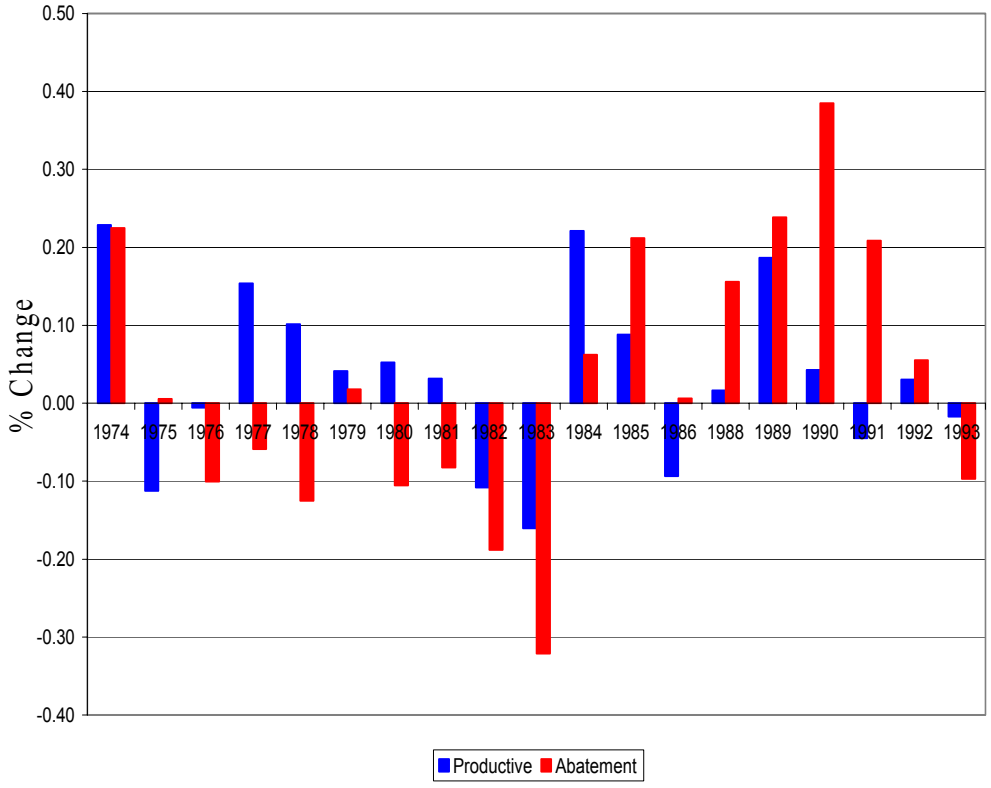


Figure 1.5: Abatement Coefficents and 95% Confidence Intervals by Industry

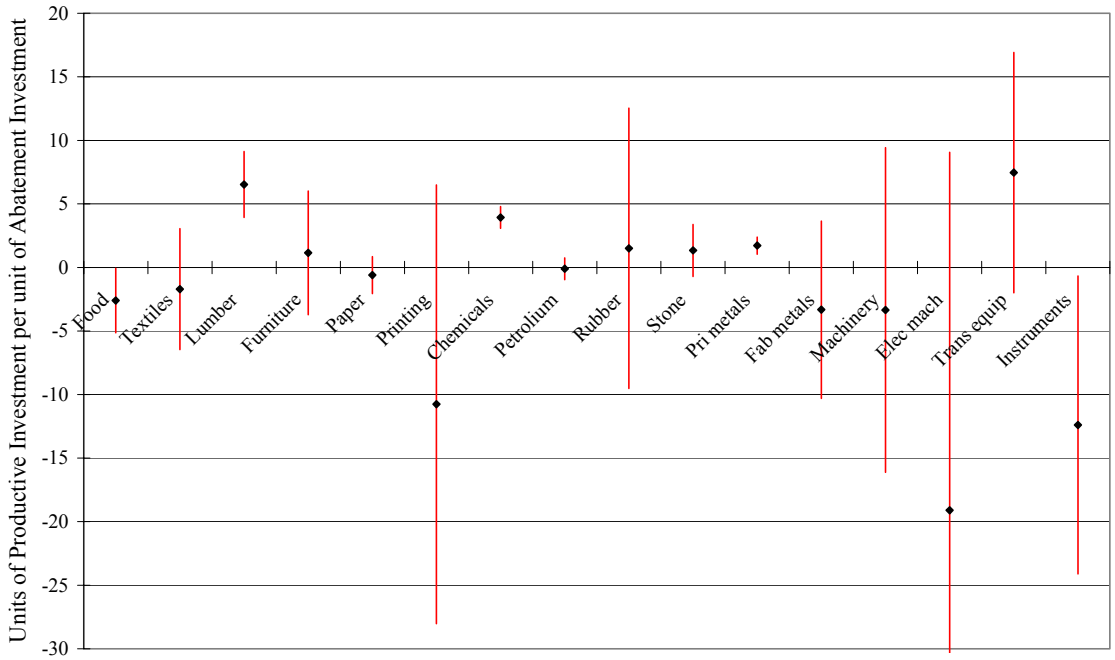


Table 1.2: Regression Results					
Coefficient	Estimate	Standard Error	Coefficient	Estimate	Standard Error
b0	1547.399 *	200.0787	**Industry Dummies**		
***Ia* Coefficient by Industry (1973-1979)**			Food	3700.91 *	441.99
Food	-2.61 *	1.29	Lumber	65.67	268.78
Textiles	-1.70	2.43	Furniture	-1019.06 *	220.43
Lumber	6.53 *	1.32	Paper	2844.62 *	632.49
Furniture	1.16	2.49	Printing	892.80 *	366.62
Paper	-0.60	0.74	Chemicals	3490.91 *	535.48
Printing	-10.76	8.81	Petroleum	2948.89 *	480.28
Chemicals	3.93 *	0.44	Rubber	-118.12	340.04
Petroleum	-0.10	0.44	Stone	1024.40 *	398.74
Rubber	1.51	5.63	Pri metals	1623.50 *	565.49
Stone	1.34	1.05	Fab metals	2524.16 *	479.97
Pri metals	1.72 *	0.34	Machinery	7105.70 *	726.82
Fab metals	-3.32	3.56	Elec mach	5181.20 *	1282.37
Machinery	-3.34	6.52	Trans equip	2983.83 *	1051.28
Elec mach	-19.10	14.37	Instruments	-478.19 *	321.10
Trans equip	7.46	4.82	**Interactive Dummies (S&P with Industry)**		
Instruments	-12.39 *	5.98	Food	6.76 *	1.57
Difference in *Ia* Coefficient Over Later Sub-period (1980 – 1993)			Textiles	1.58 *	0.65
Food	3.85 *	1.11	Lumber	-1.22	0.74
Textiles	-0.40	2.62	Furniture	0.60	0.47
Lumber	-2.06	1.60	Paper	-0.98	3.90
Furniture	1.44	4.13	Printing	2.07	1.57
Paper	4.97 *	0.90	Chemicals	-4.19	3.62
Printing	47.56 *	8.32	Petroleum	-10.11 *	2.47
Chemicals	1.98 *	0.41	Rubber	6.08 *	2.02
Petroleum	2.35 *	0.42	Stone	-1.75	1.09
Rubber	5.66	5.06	Pri metals	0.22	1.55
Stone	0.31	1.48	Fab metals	-0.01	1.44
Pri metals	1.33 *	0.48	Machinery	-12.44 *	3.20
Fab metals	2.31	2.03	Elec mach	-3.00	4.18
Machinery	22.75 *	5.38	Trans equip	2.78	3.55
Elec mach	33.33 *	5.54	Instruments	5.15 *	1.69
Trans equip	5.51	3.39	X2(63)	11657.86 *	
Instruments	25.48 *	4.74			

Figure 2.1: Stylized Growth Function of Forest Acre

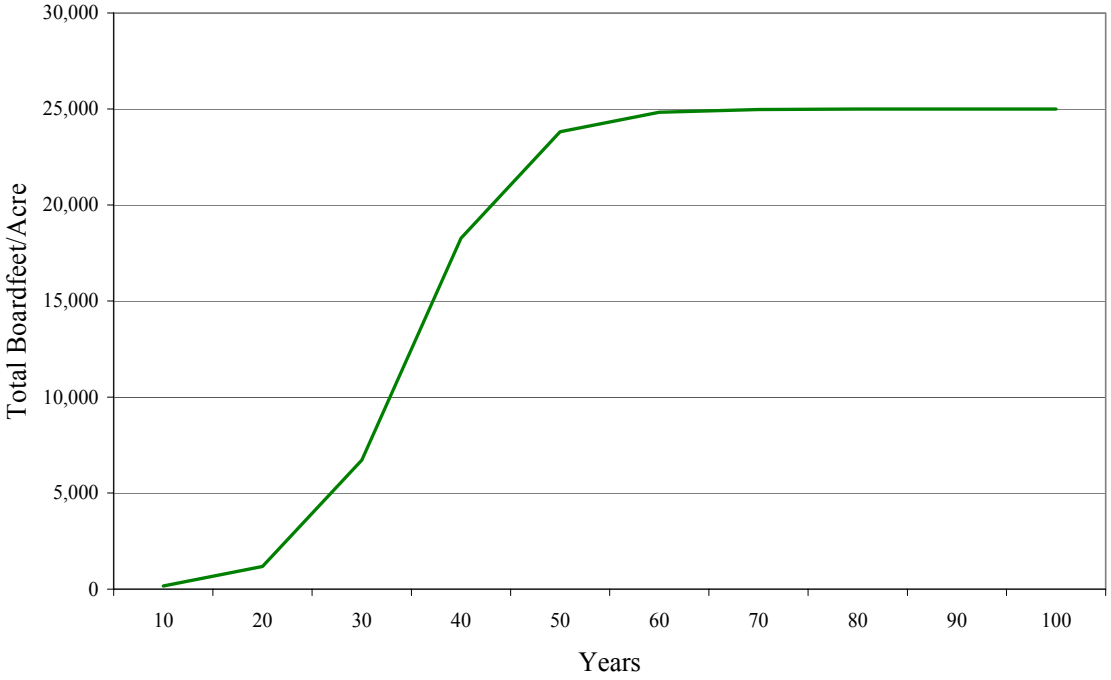


Figure 2.2: Optimal Rotation Periods and Harvest Percentages for Different Discount Rates and γ = $0.02

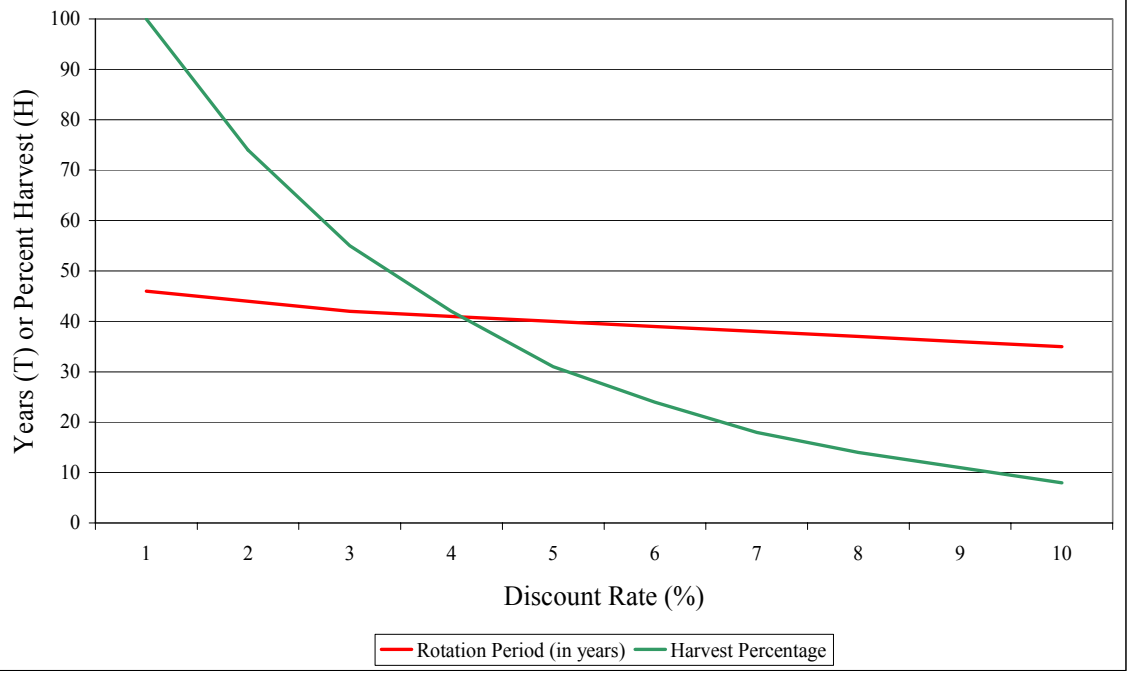


Figure 2.3: Optimal Rotation Periods and Harvest Percentages for Different Marginal Amenity Values and r = 3%

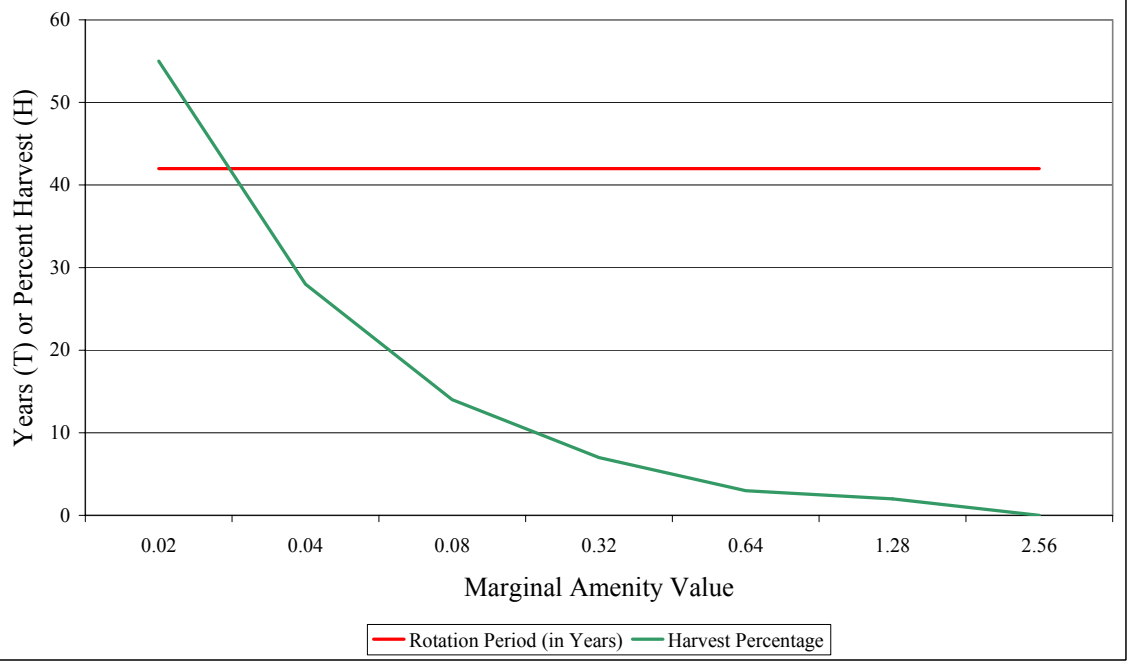


Table 2.1: Effect of r and γ On Tax Rates and Penalties			
r	τ^{cc} (rate)	$\tau^{cc}*H^2$ (penalty)	τ^{ls}
1%	$22,944	$22,944	-$10,234
2%	$29,522	$16,166	-$10,539
3%	$37,444	$11,327	-$8,891
4%	$47,014	$8,293	-$6,946
5%	$58,534	$5,625	-$5,219
6%	$72,292	$4,164	-$3,832
7%	$88,541	$2,869	-$2,771
8%	$107,449	$2,106	-$1,982
9%	$129,030	$1,561	-$1,404
10%	$153,050	$980	-$986
γ	τ^{cc} (rate)	$\tau^{cc}*H^2$ (penalty)	τ^{ls}
0.02	$37,443.75	$11,326.73	-$8,890.93
0.04	$74,887.49	$5,871.18	-$4,445.47
0.08	$149,774.98	$2,935.59	-$2,222.73
0.32	$299,549.97	$1,467.79	-$1,111.37
0.64	$599,099.94	$539.19	-$555.68
1.28	$1,198,199.87	$479.28	-$277.84
2.56	$2,396,399.74	$0.00	-$138.92

Table 3.1: Expenditure an Consumption of Goods (included in the 1996 CEX Diary Survey) Containing BPA by Consuming Women (age 18-40 years)

Good	Mean Expenditure (in $)/ 14 days	Average Price/Unit	Mean Consumption (units per day)	% of Women who Consume Zero Units on Average
Canned Food	4.03 (3.56)	0.69	0.42 (0.37)	61%
Soft Drinks	3.54 (2.63)	0.33	0.77 (0.57)	55%

Note: Number in parentheses are standard errors

Table 3.2: Average Daily Ingestion of BPA by Product

statistics produced via Monte Carlo simulation

Variable	Mean	Std. Dev.
canned food	1.24	2.40
dental sealant	5.25E-03	3.70E-04
bottled water	0.50	0.44
tupperware	4.33	4.30
canned juice	0.86	1.85
soft drinks	0.06	0.11
tap water	1.03	0.77

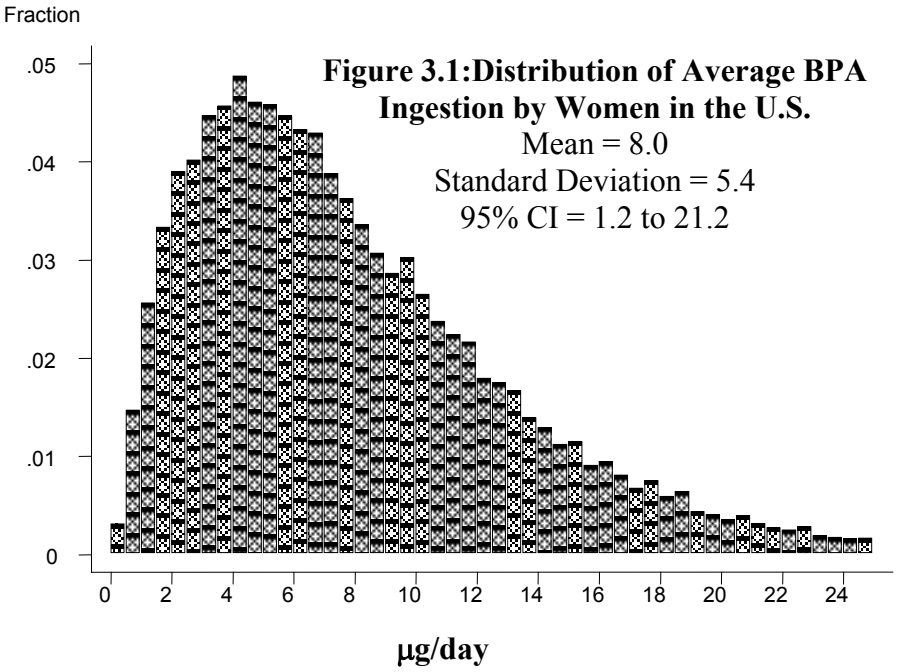


Figure 3.1:Distribution of Average BPA Ingestion by Women in the U.S.
Mean = 8.0
Standard Deviation = 5.4
95% CI = 1.2 to 21.2

Table 3.3: Dose Response Function Regression Results				
Testicular Cancer				
	Coefficient	Standard Error	95% Confidence Interval	
constant	0.0005985	0.000523	-0.0004271	0.001624
dose	3.60E-06	1.37E-07	3.34E-06	3.87E-06
dose^2	-1.54E-10	6.06E-12	-1.66E-10	-1.42E-10
Cervicovaginal Cancer				
	Coefficient	Standard Error	95% Confidence Interval	
constant	0.0005454	0.000066	0.0004161	0.0006748
dose	8.06E-08	1.54E-08	5.04E-08	1.11E-07
dose^2	-3.31E-12	6.23E-13	-4.53E-12	-2.09E-12

Figure 3.2: Dose Response Function for Cervicovaginal Cancer with Upper and Lower Bounds (95% Confidence Interval)

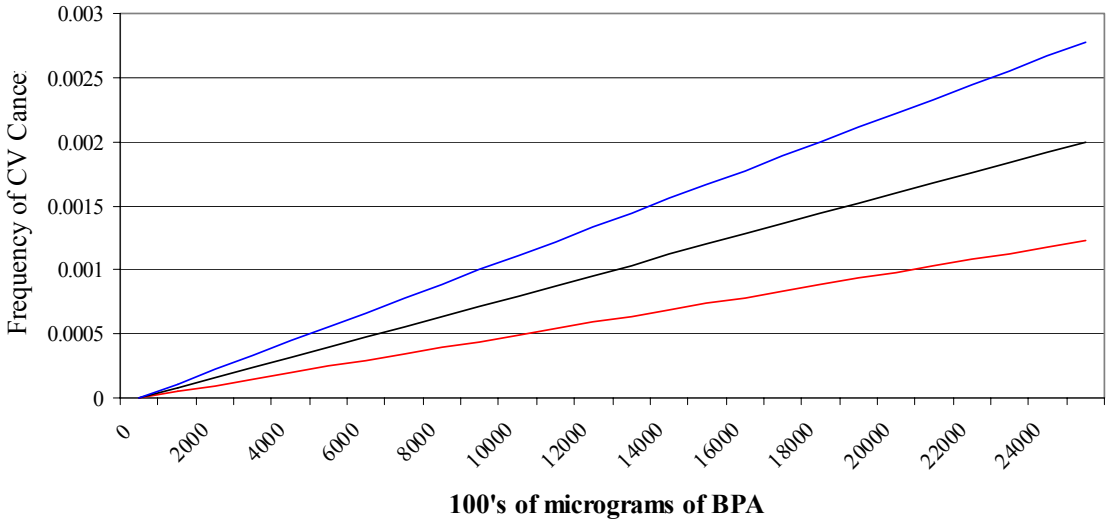


Figure 3.3: Dose Response Function (low doses) for Cervicovaginal Cancer with Upper and Lower Bounds (95% Confidence Interval)

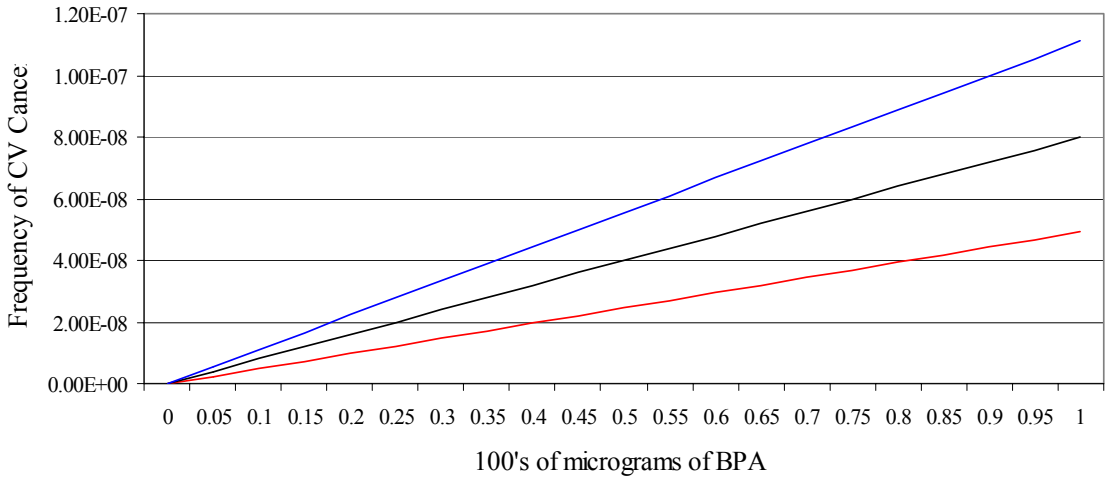


Figure 3.4: Dose Response Function for Testicular Cancer with Upper and Lower Bounds (95% Confidence Interval)

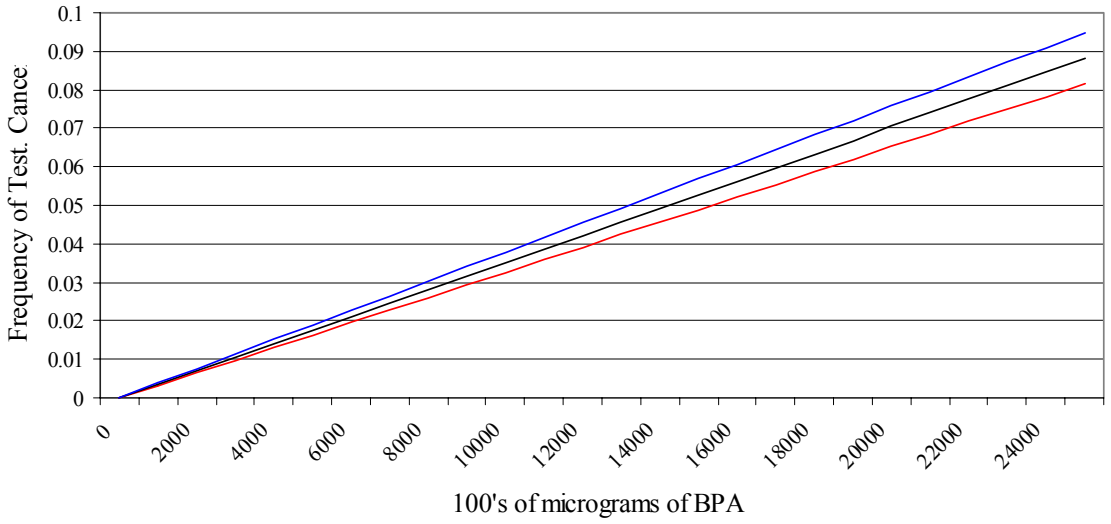
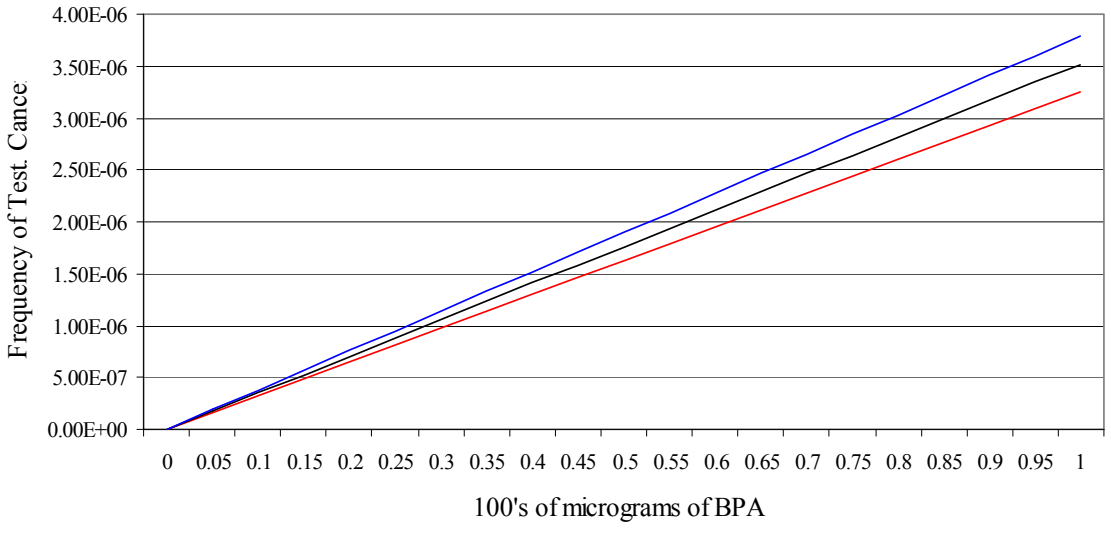


Figure 3.5: Dose Response Function (low doses) for Testicular Cancer with Upper an Lower Bounds (95% Confidence Interval)



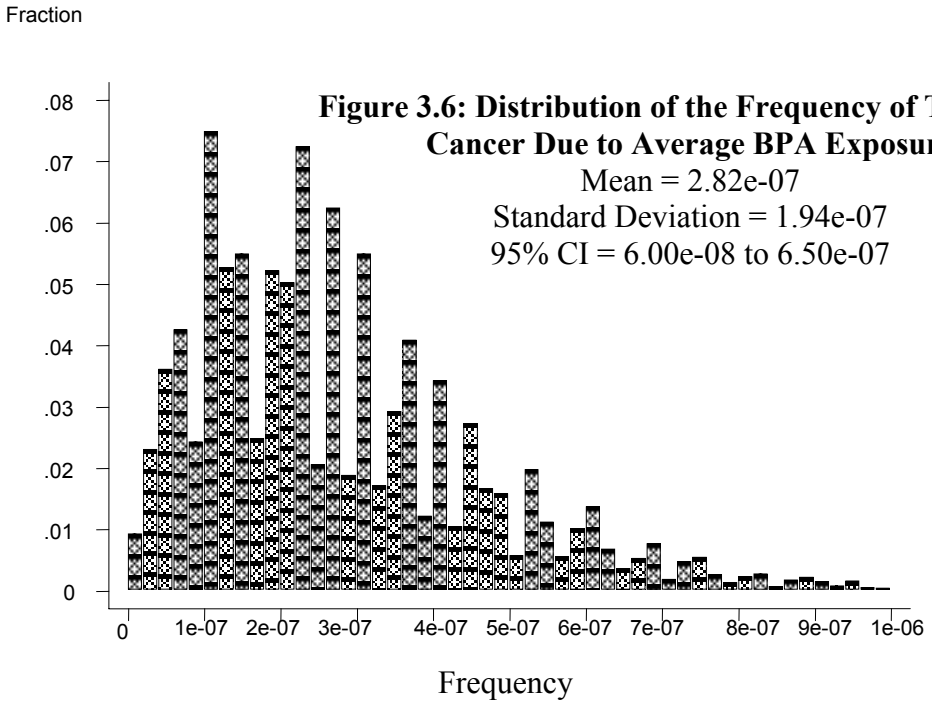


Figure 3.6: Distribution of the Frequency of Testicular Cancer Due to Average BPA Exposure

Mean = 2.82e-07

Standard Deviation = 1.94e-07

95% CI = 6.00e-08 to 6.50e-07

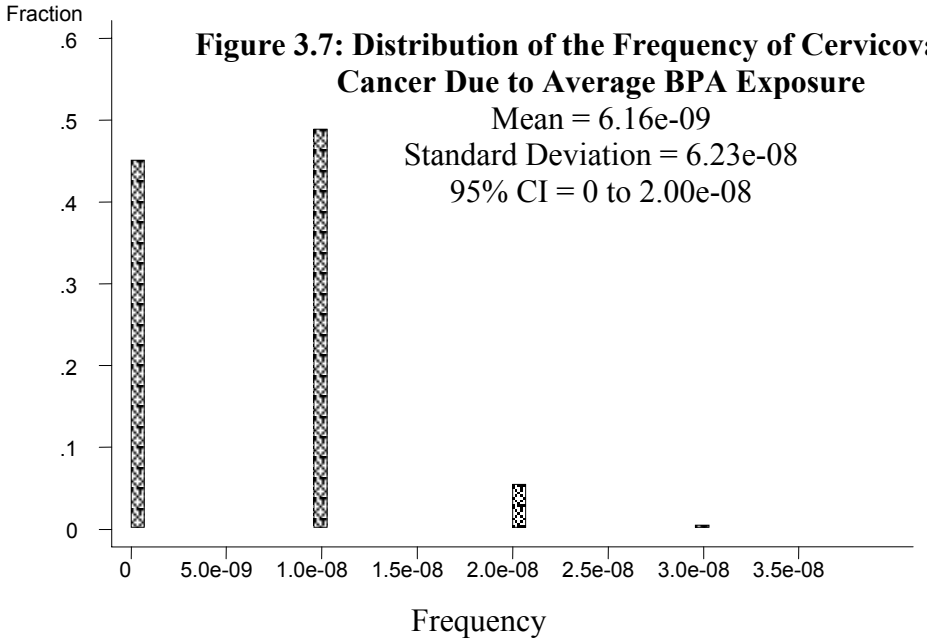


Figure 3.7: Distribution of the Frequency of Cervicovaginal Cancer Due to Average BPA Exposure

Mean = 6.16e-09

Standard Deviation = 6.23e-08

95% CI = 0 to 2.00e-08

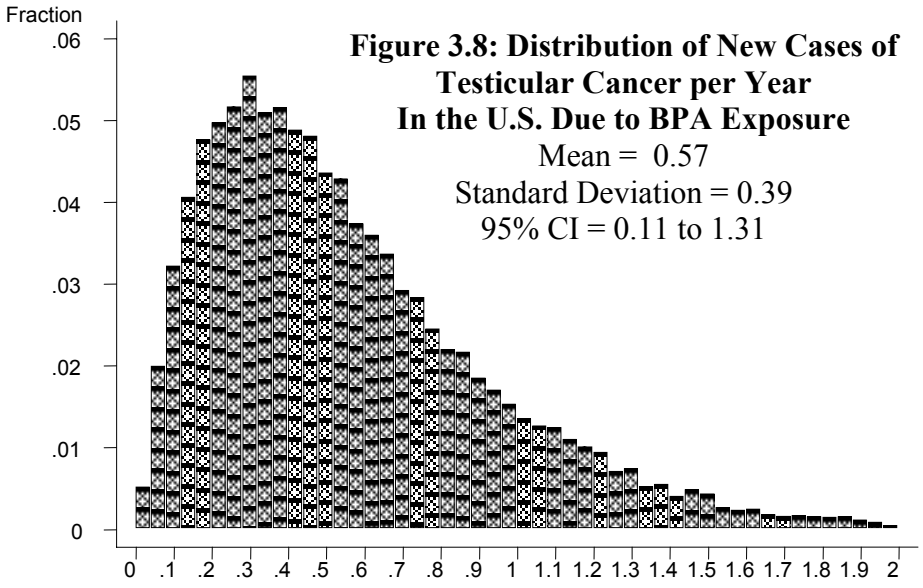


Figure 3.8: Distribution of New Cases of Testicular Cancer per Year In the U.S. Due to BPA Exposure

Mean = 0.57
Standard Deviation = 0.39
95% CI = 0.11 to 1.31

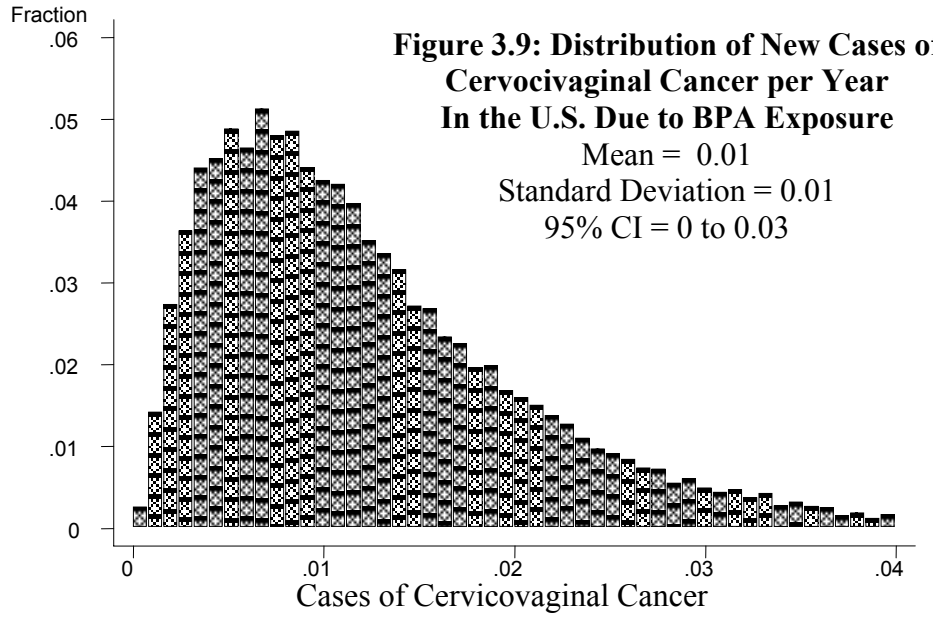


Figure 3.9: Distribution of New Cases of Cervocivaginal Cancer per Year In the U.S. Due to BPA Exposure

Mean = 0.01
Standard Deviation = 0.01
95% CI = 0 to 0.03

Table 3.4: Summary of Mortality Estimates Used in the EPA's, "The Benefits of the Clean Air Act, 1990 - 2010"

Keisner and Leeth (1991)	0.7
Smith and Gilbert (1984)	0.9
Dillingham (1985)	1.1
Butler (1983)	1.4
Miller and Guria (1991)	1.5
Moore and Guria (1988a)	3.1
Landefeld (1979)**	3.2
Viscusi, Magat, and Huber (1991b)	3.3
Gegax et. al. (1985)	4.1
Marin and Psacharopoulos (1982)	3.5
Kneiser and Leeth (1991) (Australia)	4.1
Gerking, de Haan, and Schulze (1988)	4.2
Cousineau, Lacroix, and Girard (1988)	4.5
Jones-Lee (1989)	4.7
Dillingham (1985)	4.8
Viscusi (1978, 1979)	5.1
R.S. Smith (1976)	5.7
V.K. Smith (1976)	5.8
Olson (1981)	6.4
Viscusi (1981)	8.0
R.S. Smith (1974)	8.9
Moore and Viscusi (1988a)	9.0
Kneiser and Leeth (1991) (Japan)	9.4
Herzog and Schlottman (1987)	11.3
Leigh and Folson (1984)	12.0
Leigh (1987)	12.9
Garen (1988)	16.7

** not listed in EPA report

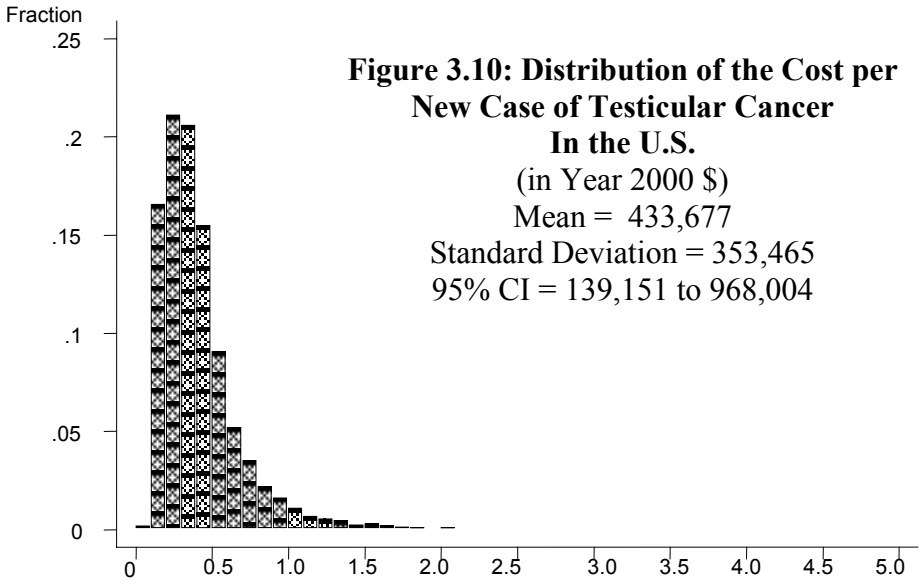


Figure 3.10: Distribution of the Cost per New Case of Testicular Cancer In the U.S.
(in Year 2000 $)
Mean = 433,677
Standard Deviation = 353,465
95% CI = 139,151 to 968,004

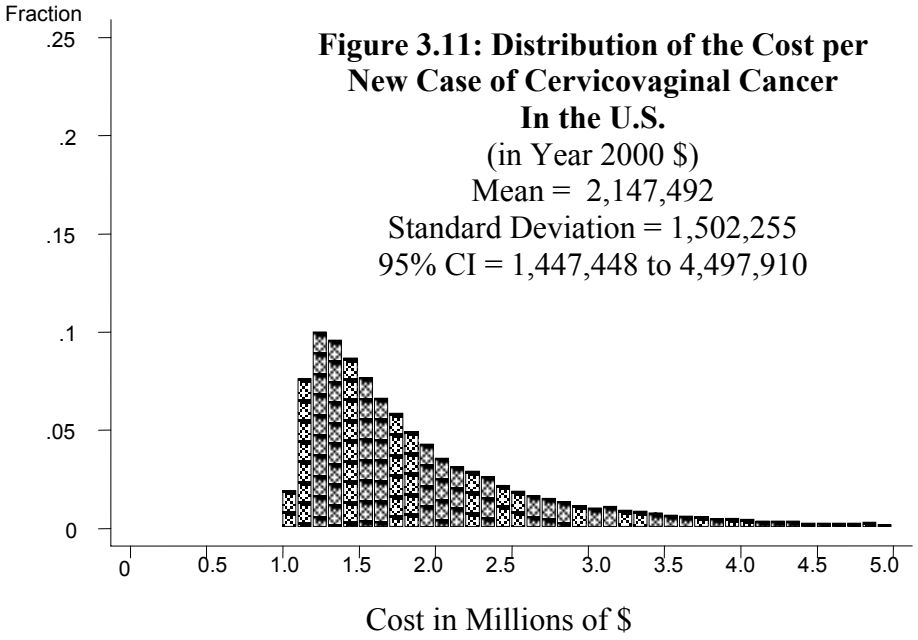


Figure 3.11: Distribution of the Cost per New Case of Cervicovaginal Cancer In the U.S.
(in Year 2000 $)
Mean = 2,147,492
Standard Deviation = 1,502,255
95% CI = 1,447,448 to 4,497,910

Table 3.6: Results of Simulation on Damages Due to BPA Exposure (in year 2000 $)				
	Total Costs, All Time, Discounted			
	Mean	Standard Deviation	95% Confidence Interval	
Total	332,377	690,776	11,347	1,304,023
Testicular Cancer	261,538	633,165	2167	1,114,465
Cervicovaginal Cancer	70,838	136,152	1919	259,614
	Annual Costs			
	Mean	Standard Deviation	95% Confidence Interval	
Total	271,791	294,349	43,808	748,139
Testicular Cancer	244,573	277,575	30,980	694,868
Cervicovaginal Cancer	27,218	31,204	4221	73,741

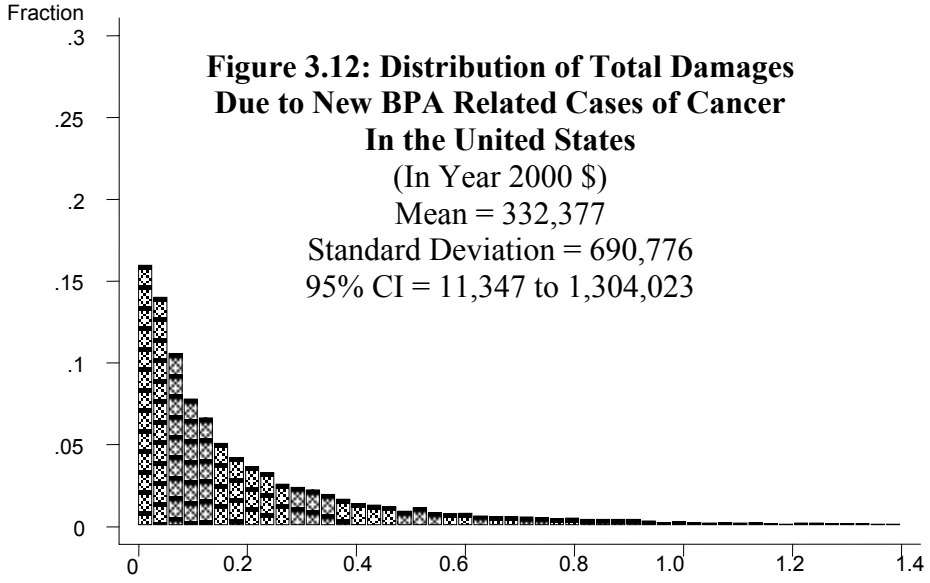


Figure 3.12: Distribution of Total Damages Due to New BPA Related Cases of Cancer In the United States
(In Year 2000 $)
Mean = 332,377
Standard Deviation = 690,776
95% CI = 11,347 to 1,304,023

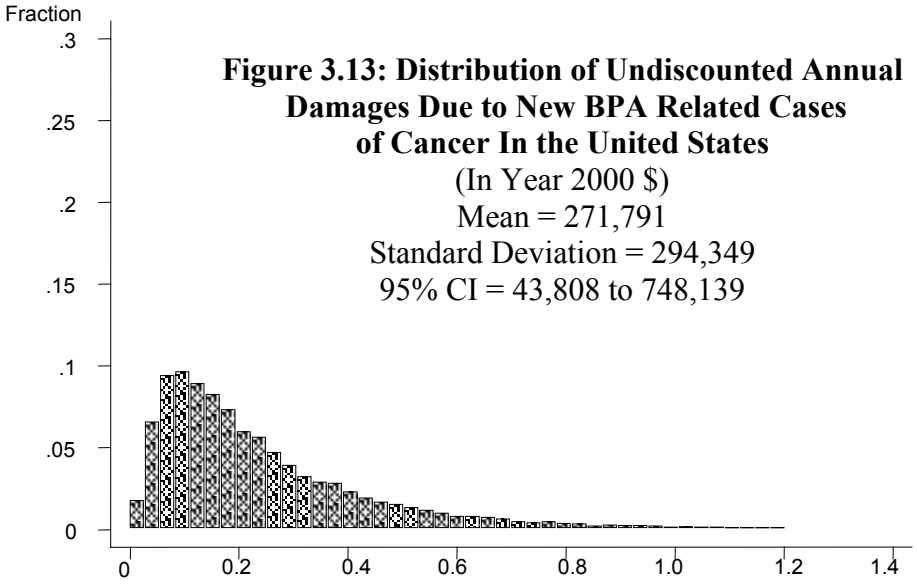


Figure 3.13: Distribution of Undiscounted Annual Damages Due to New BPA Related Cases of Cancer In the United States
(In Year 2000 $)
Mean = 271,791
Standard Deviation = 294,349
95% CI = 43,808 to 748,139

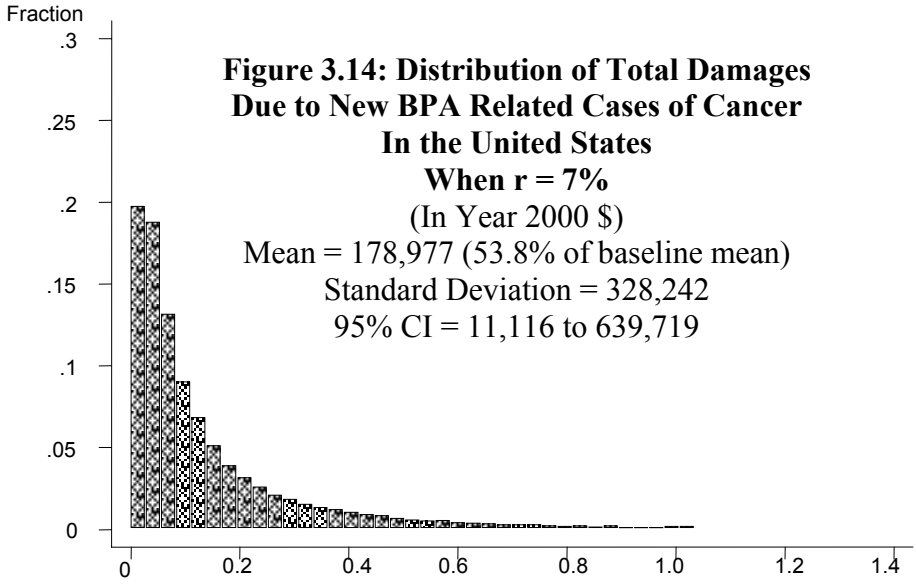


Figure 3.14: Distribution of Total Damages Due to New BPA Related Cases of Cancer In the United States When r = 7%
(In Year 2000 $)
Mean = 178,977 (53.8% of baseline mean)
Standard Deviation = 328,242
95% CI = 11,116 to 639,719

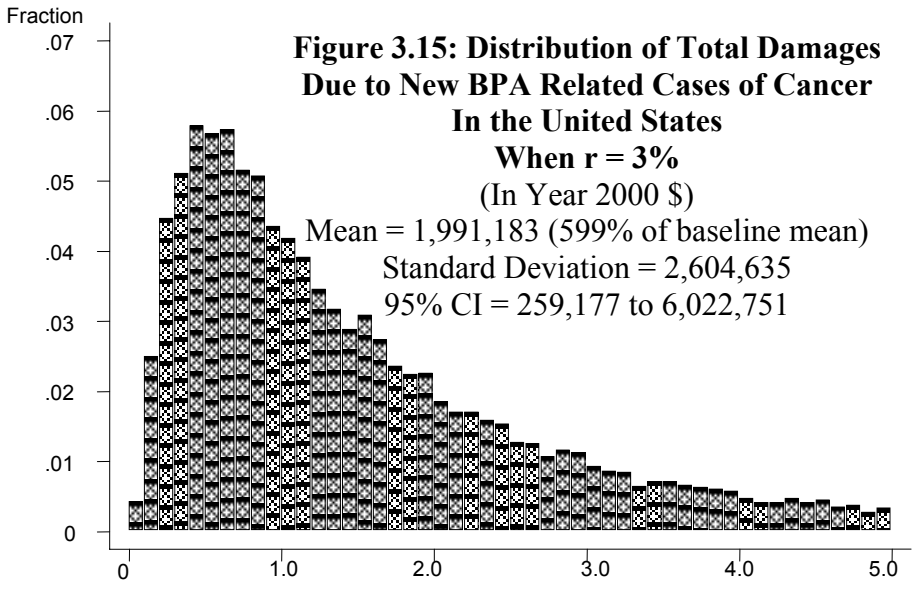


Figure 3.15: Distribution of Total Damages Due to New BPA Related Cases of Cancer In the United States When r = 3%
(In Year 2000 $)
Mean = 1,991,183 (599% of baseline mean)
Standard Deviation = 2,604,635
95% CI = 259,177 to 6,022,751

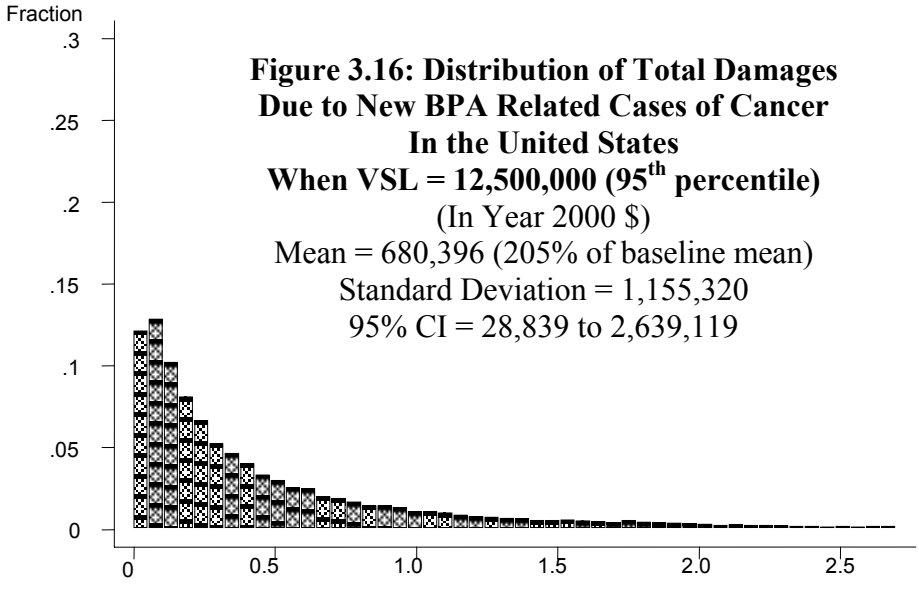


Figure 3.16: Distribution of Total Damages Due to New BPA Related Cases of Cancer In the United States When VSL = 12,500,000 (95th percentile)
(In Year 2000 $)
Mean = 680,396 (205% of baseline mean)
Standard Deviation = 1,155,320
95% CI = 28,839 to 2,639,119

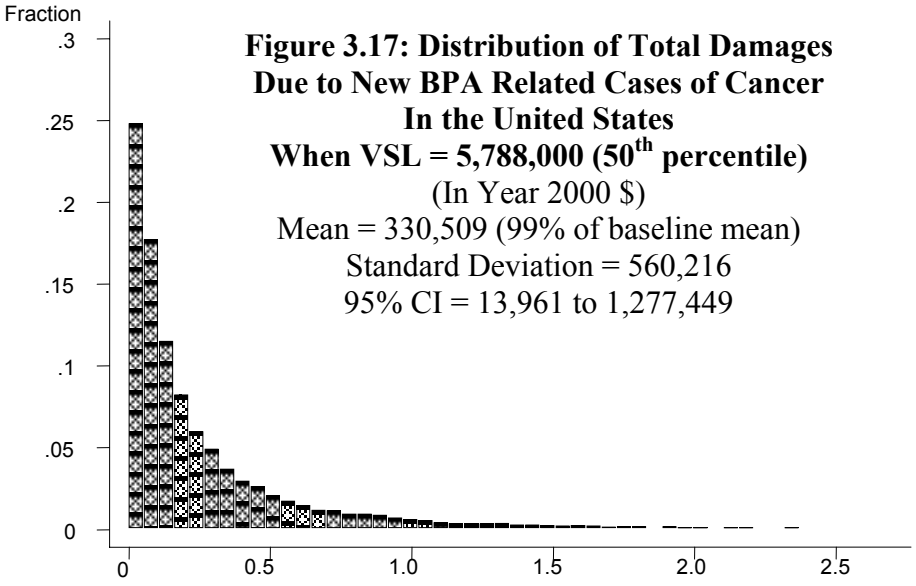


Figure 3.17: Distribution of Total Damages Due to New BPA Related Cases of Cancer In the United States When VSL = 5,788,000 (50th percentile)
(In Year 2000 $)
Mean = 330,509 (99% of baseline mean)
Standard Deviation = 560,216
95% CI = 13,961 to 1,277,449

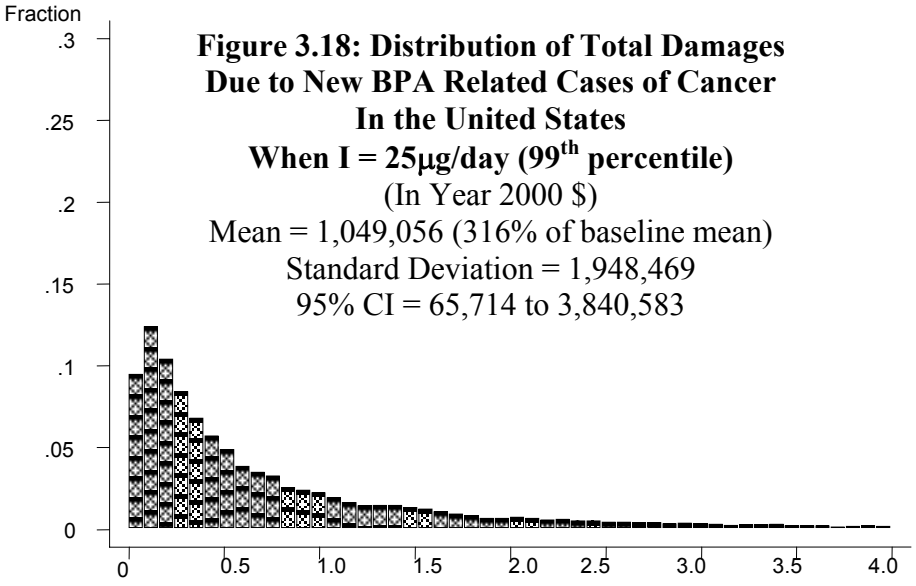


Figure 3.18: Distribution of Total Damages Due to New BPA Related Cases of Cancer In the United States When I = 25μg/day (99th percentile)
(In Year 2000 $)
Mean = 1,049,056 (316% of baseline mean)
Standard Deviation = 1,948,469
95% CI = 65,714 to 3,840,583

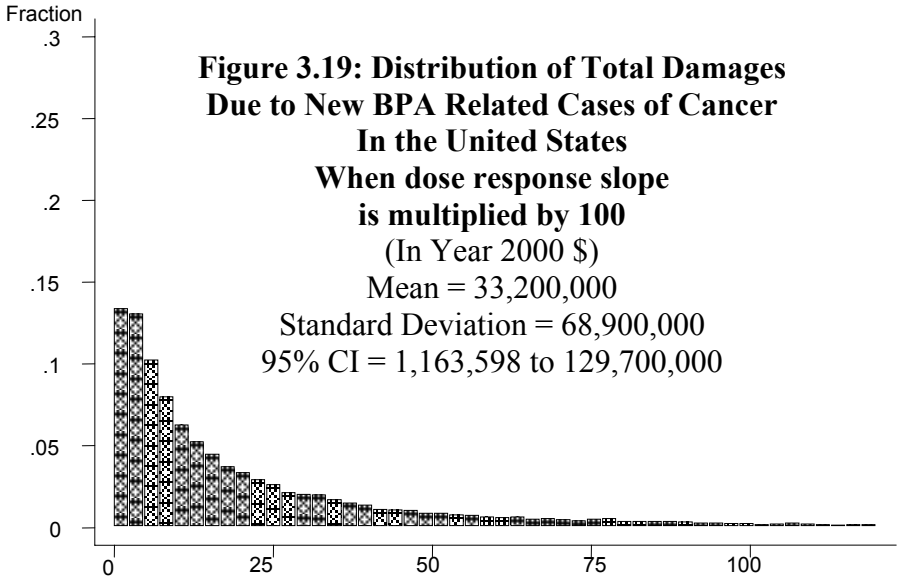


Figure 3.19: Distribution of Total Damages Due to New BPA Related Cases of Cancer In the United States When dose response slope is multiplied by 100
(In Year 2000 $)
Mean = 33,200,000
Standard Deviation = 68,900,000
95% CI = 1,163,598 to 129,700,000

Table 3.7: BPA Leached into Canned Food (Brotons et al., 1995)

Food	mL of liquid	Mean BPA in Food (μg/can)	Standard Deviation
Peas	50	22.9	8.8
Artichokes	150	18.6	6.5
Green beans	190	11.9	5.3
Mixed vegetables	220	10.1	4.3
Corn	15	4.5	2.6
Mushrooms	145	4.2	4.1
Asparagus	80	Not Detectable	-
Palm hearts	280	Not Detectable	-
Peppers	140	Not Detectable	-
Tomatoes	140	Not Detectable	-

Appendices

Appendix A

Deriving the First Order Conditions for the General Model of Investment Over n-types of Capital

The model in Part 2 of the Chapter 1 is an optimal control problem, so the first order conditions can be found by differentiating the following Hamiltonian:

(A1) $$H = \{\pi(K, I, W) - P_k I\}e^{-R(t)} + \Lambda(I - \delta K)$$

This yields the following first order conditions:

(A2) $$H_I \Rightarrow (\nabla_I \pi - P_k)e^{-R(t)} + \Lambda = 0$$

(A3) $$H_K \Rightarrow (\nabla_K \pi)e^{-R(t)} - \Lambda\delta = -\dot{\Lambda}$$

(A4) $$H_\Lambda \Rightarrow \dot{K} = I - \delta K$$

Making use of the current value transormation of Λ, the first order conditions are rewritten in the following manner:

(A5) $$\lambda = P_k - \nabla_I \pi$$

(A6) $$\dot{\lambda} - \lambda(rJ + \delta) = -\nabla_K \pi$$

(A7) $$\dot{K} = I - \delta K$$

To better interpret these first order conditions, (A6) is integrated along the optimal paths of I and K. This yields:

(A8) $$\lambda(\infty)e^{-(R(\infty)J+\delta\infty)} - \lambda(0) = -\int_0^{\infty} \nabla_K \pi e^{-(R(t)J+\delta t)}$$

To simplify further, a set of transversality conditions is used:

(A9) $$\forall j, \ldots \lim_{t\to\infty} \lambda_j(t) e^{-(R(t)-\delta_{jj}t)} = 0$$

Applying (ix) to (viii), the FOC's can now be rewritten as they appear in Section 2 of Chapter 1.

Appendix B

Differentiation of $\phi(I)$ With Respect to I_p

Assume $\phi(I)$ takes the quadratic form:

(B1) $$\phi(I) = \alpha + \gamma' I + I' \Gamma I$$

Where:

(B2) $$I = \begin{bmatrix} I_p \\ I_a \end{bmatrix}$$ (B3) $$\gamma' = \begin{bmatrix} \gamma_p & \gamma_a \end{bmatrix}$$ (B4) $$\Gamma = \begin{bmatrix} \Gamma_{pp} & \Gamma_{pa} \\ \Gamma_{ap} & \Gamma_{aa} \end{bmatrix}$$

Thus:

(B5) $$\phi(I) = \alpha + \begin{bmatrix} \gamma_p & \gamma_a \end{bmatrix} \begin{bmatrix} I_p \\ I_a \end{bmatrix} + \begin{bmatrix} I_p & I_a \end{bmatrix} \begin{bmatrix} \Gamma_{pp} & \Gamma_{pa} \\ \Gamma_{ap} & \Gamma_{aa} \end{bmatrix} \begin{bmatrix} I_p \\ I_a \end{bmatrix}$$

Multiplying (B5) out gives

(B6) $$\phi(I) = \alpha + \gamma_p I_p + \gamma_a I_a + \Gamma_{pp} I_p^2 + 2\Gamma_{ap} I_p I_a + \Gamma_{aa} I_a^2$$

Taking the partial derivative with respect to I_p yields the expression for $\partial\phi(I)/\partial I_p$ found in Section 3 of the Chapter I.

Appendix C

Additional Demographic Groups Potentially Affected by Bisphenol-A

There is no question that the fetus is uniquely vulnerable to disturbances in estrogen levels. However, some studies show that additional groups may also be susceptible, albeit less so, to adverse outcomes due to BPA exposure. For example, rapidly developing neonates, exposed to BPA in some baby bottles, may be vulnerable. Neonatal estrogen treatment has been shown to cause deformities and precancerous conditions in female mice and rats (Forsberg, 1979, Bern *et. al.*, 1987 Medlock, *et. al.*, 1988, Brody and Cunha, 1989). Furthermore, though the National Research Council (1999) states that the results of exposure to BPA as an adult are at worst transitory, there is evidence linking adult estrogen treatment to endometrial cancer (Ziel, *et. al*., 1975). Adult exposure to exogenous estrogen may also promote malignant breast tumor growth (Dickson, *et. al.,* 1986).

Appendix D

Additional Adverse Outcomes Associated with Bisphenol-A

Cancers known to be caused by prenatal exogenous estrogen exposure were chosen as the focus of this study due to data considerations and their relative importance and cost. However, Bisphenol-A also may lead to infertility through reduced sperm production or genital tract deformation. It causes increased prostate size of male offspring when environmentally relevant doses are fed to pregnant female mice (Nagel, *et. al.*, 1997). It also increases testis weight and reduces daily sperm production in male offspring of pregnant mice fed BPA (vom Saal *et. al.*, 1998). Bisphenol-A induces hyperprolactimia in some genetically predisposed rats (Steinmetz *et. al*, 1997). BPA is also linked to abnormalities in behavior and sexuality in mice (vom Saal, *et. al.*, 1998).

Studies using other estrogen-like chemicals link increased hormone levels at critical periods of fetal development to female genital tract deformation, such as lesions on and malformation of the uterus, cervix, ovaries, and vagina, as well as infertility and premature birth in future generations (Herbst *et. al.*, 1971, Forsberg, 1979, McLachlan, 1979, Barnes *et. al.*, 1980, Cousins *et. al.*, 1980, Newbold *et. al.*, 1982, Thorpe *et. al.*, 1990, Greco *et. al.*, 1993). Prenatal exogenous estrogen exposure is also linked prostate enlargement, cryporchisdism, hypospadias, and reduced sperm count and efficiency in humans (Bullock, *et. al.*, 1988, vom Saal, *et. al.*, 1997, vom Saal, *et. al.*, 1998).

It is also possible that BPA exposure may be linked other types of cancer, but at this point, no hard evidence has been produced.

Appendix E

Calibration of Leaching Parameters

For canned food, data from Brotons, *et. al.* (1998), shown in Table 3-E1 is used. Since consumption of a particular type of canned good is difficult to untangle using existing data, the leaching data from Brotons is averaged. For peas, artichokes, green beans, mixed vegetables, corn, and mushrooms, the leaching distribution is assumed to be lognormal. For canned food where the amount leached into food is undetectable, asparagus, palm hearts, peppers, and tomatoes, the leaching distribution is assumed to be uniform between 0 and the detection limit of 5 μg/mL. In each round of 25,000 draws, draws are taken from each distribution individually and summed. This number is divided by 10 to provide the average amount leached from an average can of food.

For dental sealant, data from Olea, *et. al.* (1996) is used. Olea finds between 89.8 and 931 μg of BPA in the saliva of patients one hour after the application of 50 mg of sealant. However, Fung, *et. al.* (2000) points out that a typical tooth only requires 8 mg of sealant. Therefore the upper and lower bounds on BPA leaching as estimated by Olea are scaled by a factor of 6.25. Thus a uniform distribution ranging from 14.37 μg/tooth to 148.96 μg/tooth is used.

To calibrate the rest of the leaching parameters, data from Takao, *et. al.* (1999) is used. Takao finds less than 1 ppb in autoclaved soft drink cans. Under the assumption that soft drink can holds 12 fluid ounces, 1 ppb is converted to micrograms to form the upper

bound of a uniform distribution ranging between 0 μg/can and 0.34 μg/can leached. Bottled water is assumed to have the same leaching rate as the 0 to 6.5 ppb range detected in polycarbonate baby bottles. The range is scaled to calculate the uniform range (0 μg to 1.48 μg) that could be found in an 8 ounce serving. Tap water is assumed to have the same amount of BPA as the 0 to 2.4 ppb range found in river water samples. The uniform range of 0 μg to 0.54 μg that is used in the model is calculated by again scaling to an 8 ounce serving. Plastic ware is also assumed to have the same leaching rate detected in polycarbonate baby bottles. The uniform range of 0 μg to 23.4 μg per container is calculated by scaling the polycarbonate bottle data to represent the amount that could be found in a container ranging in size between 0.23 cups to 15 cups. Containers of any size in this range are assumed to have an equal likelihood of being used at any given time. Canned fruit juice can come in containers resembling food cans or soft drink cans. Therefore leaching from canned juice is assumed to be the average of the amount leached from a canned food and the amount leached from a soft drink can.

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